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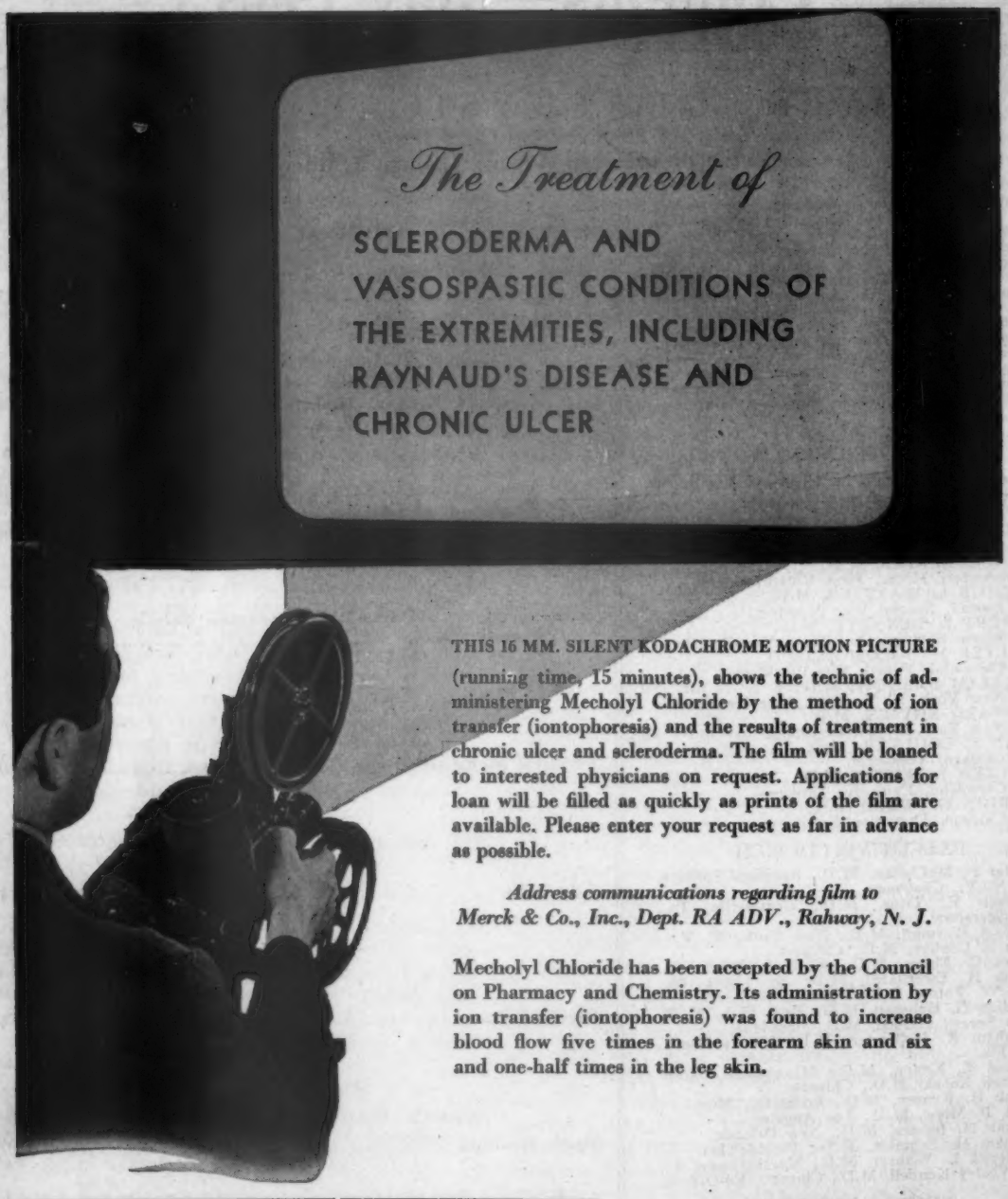
Printed in U.S.A.



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TECHNICS OF PROGRESSIVE RESISTANCE EXERCISE *

THOMAS L. DE LORME, M.D.

and

ARTHUR L. WATKINS, M.D.

BOSTON

During the past three years many improvements have been made in the technics of administering progressive resistance exercises. This purely technical paper has been prepared to explain the new technics and the improvements on the old.[1] No reference will be made to the physiologic aspects of exercise, indications for use or results obtained. However, the methods of performing progressive resistance exercises are considered in detail.

When the exercises originally described were designated as "heavy resistance exercises," this name was not intended to imply that only great poundages are used and that a muscle initially must have nearly normal power. The resistance is "heavy" only when the amount of muscle power is taken into consideration. For instance, a few ounces would be as heavy for a severely weakened muscle in a patient with poliomyelitis as 50 or 100 pounds would be to a normal muscle. Regardless, it has become apparent that the term "heavy resistance exercises" bears false implications, and the term "progressive resistance exercises" was suggested as being more appropriate. This term has been adopted, and no further reference will be made to the original name.

Recent developments have made it imperative to revise, define and standardize the terminology used in progressive resistance exercise. Every attempt has been made to develop simple, logical and self-explanatory terms.

The principle of counterbalancing has been employed for exercising muscles so weak that they cannot complete a full arc of motion against gravity By a system of pulleys and a counterbalancing load, enough of the weight of the extremity is offset to permit the weak muscle to carry the extremity through a full range of motion. This method makes it possible to give progressive exercises through a full arc of motion to extremely weak muscles and is a substantial improvement over the method previously described — that of overloading the weak muscle through a limited range of motion.[1] The muscle must still exert its maximum power, even though it is assisted by a counterbalance. Therefore, the principles of progressive resistance exercise are not violated by counterbalancing.

Before entering upon the discussion of counterbalancing in exercise, two terms must be defined: One is "exercise load"; the other, "muscle load." The exercise load is the load with which the exercise is performed and does not necessarily refer to the resistance the muscle must overcome during the exercise. As will be explained later, this load may serve to assist (fig. 2) or resist (fig. 3) the muscle during exercise. The muscle load is the load (or resistance) which the muscle must actually overcome during the exercise. For stronger muscles, the muscle load includes both the weight of the extremity and the exercise load (fig. 3). For weak muscles, the muscle load is only a portion of the weight of the extremity, the remainder having been offset by the exercise load serving as a counterbalance.

* From the Department of Physical Medicine, Massachusetts General Hospital.
* Aided by a grant from The Pope Foundation, Inc.

1. DeLorme, T. L.: Restoration of Muscle Power by Heavy Resistance Exercises, J. Bone & Joint Surg. 27:645 (Oct.) 1945; Heavy Resistance Exercises, Arch. Phys. Med. 27:607 (Oct.) 1946.

The exercise load is used in counterbalancing to offset part of the weight of the extremity in case of extremely weak muscles (fig. 2); for stronger muscles, the exercise load is added to the weight of the extremity (fig. 3). Thus, when counterbalancing is used, the exercise load assists the muscle — i. e., reduces the muscle load — whereas for stronger muscles the exercise load resists the muscle. Further consideration shows that when the exercise load is used as a counterbalance, its weight never exceeds the weight of the extremity, for if it did the exercise would become purely passive, since the exercise load would be lifting the entire weight of the extremity.

For the muscle possessing more than enough strength to complete an arc of motion against gravity, the weight of the extremity alone does not offer sufficient resistance to stimulate hypertrophy. Therefore the exercise load is added to the weight of the extremity.

The term "load-assisting exercises" applies to those in which the exercise load assists the muscle (fig. 2). The term "load-resisting exercises" refers to those in which the exercise load resists the muscle (fig. 3). It should be remembered that the loads referred to here are the exercise loads, not the muscle loads.

In the load-resisting exercises the exercise load increases are based on the 10 repetition maximum[2] exactly as originally described,[1] and as muscle power increases so does the 10 repetition maximum increase. However, in the load-assisting exercises the exercise loads used are based on the 10 repetition minimum. The extremely weak muscle must be assisted by the exercise load if it is to lift the extremity. If there is too much assistance (i. e., exercise load too heavy), the muscle may perform more than 10 repetitions, and if too little assistance is rendered (i. e., exercise load too light) 10 repetitions are not possible. There is an exercise load which barely makes possible 10 repetitions. This load is called the "10 repetition minimum" and is the basis for increasing the muscle loads in load-assisting exercises.

In view of the preceding discussion, it follows that in load-resisting exercises, the muscle load increases with increases in the exercise load, but in the load-assisting exercises the opposite is true. Here the muscle load increases with decreases in the exercise load.

In the initial publications concerning progressive resistance exercise, 70 to 100 repetitions were advocated, the repetitions being performed in 7 to 10 sets with 10 repetitions per set. Further experience has shown this figure to be too high and that in most cases a total of 20 to 30 repetitions is far more satisfactory. Fewer repetitions permit exercise with heavier muscle loads, thereby yielding greater and more rapid muscle hypertrophy.

By use of small muscle loads initially and then increasing them after each set of 10 repetitions, the muscle is "warmed up" preparatory to exerting its maximum power for 10 repetitions. The following method for determining the approximate exercise load for each set of 10 repetitions for load-resisting exercises and load-assisting exercises has proved practical and effective:

Load-Resisting Exercises

First set of 10 repetitions	Use ½ of 10 repetition maximum
Second set of 10 repetitions	Use ¾ of 10 repetition maximum
Third set of 10 repetitions	Use 10 repetition maximum

Load-Assisting Exercises

First set of 10 repetitions	Use twice the 10 repetition minimum
Second set of 10 repetitions	Use 1½ times the 10 repetition minimum
Third set of 10 repetitions	Use 10 repetition minimum

2. The 10 repetition maximum is the most weight that can be lifted correctly through a full arc of motion for 10 repetitions.

For example, let us suppose that the patient with poliomyelitis in figure 2 cannot abduct against gravity because of severe deltoid involvement. This means that the weight of the arm is too much for the deltoid to lift. We know then that if the deltoid is to abduct the arm, some of the weight of the arm must be eliminated. This is accomplished by counterbalancing. We determine the 10 repetition minimum by starting with a counterbalancing load that makes abduction possible for the weak deltoid for more than 10 repetitions. Then, by gradually reducing the counterbalancing load, we find the least assistance the deltoid must have in order to perform 10 repetitions. Let us suppose this is 10 pounds. Then, for the

First set of 10 repetitions	Use 20 pounds of counterbalance
Second set of 10 repetitions	Use 15 pounds of counterbalance
Third set of 10 repetitions	Use 10 repetition minimum (10 pounds)

In this way the resistance the muscle must overcome increases after each 10 repetitions, thus permitting it to "warm up."

The position assumed for the performance of certain exercises makes counterbalancing unnecessary for the extremely weak muscles (fig. 5 or 29), since the dependent position of the extremity in the initial phase of the exercise permits gravity to pull it through a large range of motion without the help of muscle power. In these exercises the pull of gravity eliminates the weight of the extremity as a factor in the exercise. Therefore these exercises are treated as load-resisting exercises and the exercise load increases are based on the 10 repetition maximum.

The so-called Complete Exercise Unit has been used for demonstrating the progressive resistance exercise technics (fig. 1).

Shoulder and Arm Exercises

A. Shoulder Abduction. — The load-assisting exercise is shown in figure 2. Approximately 125 degrees of counterbalanced abduction is possible by this arrangement. If it is desirable to perform this exercise while standing, the same pulley can be used (fig. 13). The load-resisting abduction exercise is illustrated in figure 3. Here the weight is held in the hand.

B. Shoulder Adduction. — The load-assisting exercise shown in figure 4. The load-resisting exercises may be performed in the same position and holding a dumbbell in the hand. This exercise may also be performed in the position shown in figure 5, in which case counterbalancing is not necessary regardless of how weak the muscle, since gravity will pull the arm into full adduction without muscle aid.

C. Forward Flexion. — Forward flexion exercises are shown in figures 6 and 7.

D. Shoulder Rotation. — The arrangement shown in figure 8 may be used for load-assisting external rotation or load-resisting internal rotation. Load-resisting external rotation is performed as shown in figure 9. It is obvious that no counterbalancing is necessary in internal rotation, since the arm falls into internal rotation because of the pull of gravity (fig. 9).

E. Shoulder Elevation. — The shoulder is depressed inferiorly; then an effort is made to touch ears with the shoulder, thus completing a shrugging motion (fig. 10).

F. Elbow Extension and Flexion. — The position demonstrated in figure 11 may be employed for load-assisting biceps exercises or load-resisting triceps exercises. This position, owing to the pull of gravity, makes a load-assisting triceps exercise unnecessary. The strap may be used as shown to help prevent substitution. Load-resisting biceps exercises are shown in figure 12.

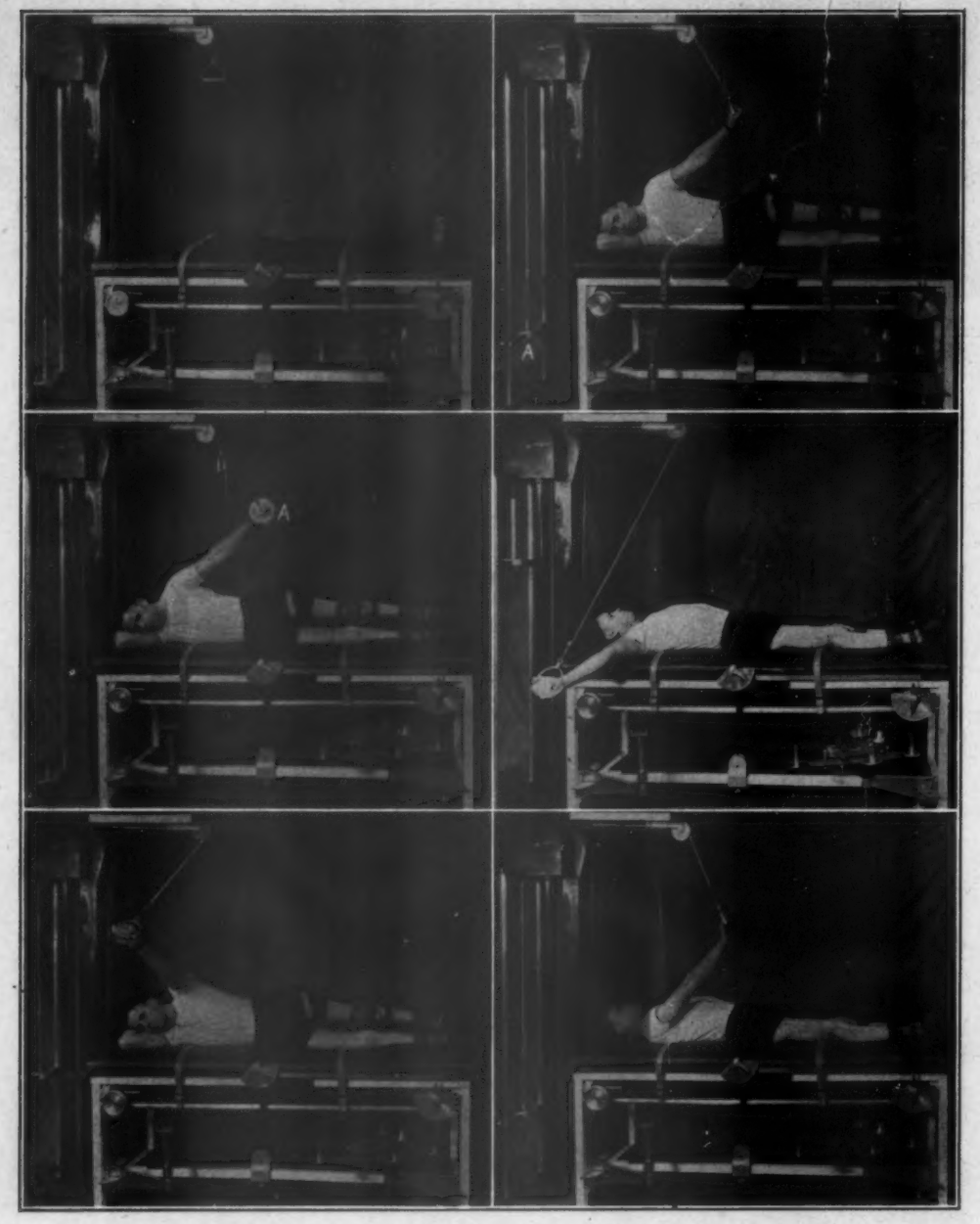

Fig. 1 (top left). — The Complete Exercise Unit.

Fig. 2 (top right). — Load-assisting exercise; shoulder abduction. The exercise load, *A*, serves as a counterbalance and assists the deltoid in abduction. The muscle load here is that portion of the weight of the extremity not eliminated by the exercise load.

Fig. 3 (center left). — Load-resisting exercise; shoulder abduction. The combined weight of the exercise load, *A*, and the weight of the extremity make up the muscle load.

Fig. 4 (center right). — Load-resisting exercise; shoulder abduction. If because of biceps or triceps weakness the elbow cannot remain locked in extension during performance of this exercise, a light splint may be secured along the posterior aspect of the arm to prevent elbow motion.

Fig. 5 (bottom left). — Load-resisting exercise; shoulder adduction. Counterbalancing is not needed, since gravity assists the exercise.

Fig. 6 (bottom right). — Load-assisting exercise; shoulder flexion.

Most of these shoulder and arm exercises may be performed in weight-bearing position by use of the overhead pulley and small dumbbells (fig. 13).

Fig. 7 (top left). — Load-resisting forward flexion exercise.

Fig. 8 (top right). — Load-resisting internal rotation exercise, or load-assisting external rotation exercise.

Fig. 9 (center left). — Load-resisting external rotation exercise.

Fig. 10 (center right). — Load-resisting shoulder girdle elevation exercise.

Fig. 11 (bottom left). — Load-assisting biceps exercise, or load-resisting triceps exercise. This position eliminates the necessity of a load-assisting triceps exercise because of the pull of gravity.

Fig. 12 (bottom right). — Load-resisting biceps exercise.

Hyperextension Exercises for the Spine

If weakness of the back muscles makes it impossible to raise the trunk off the table, counterbalancing as shown in figure 14 is used. In load-resisting hyperextension exercises, the load is added to a leather weight pan

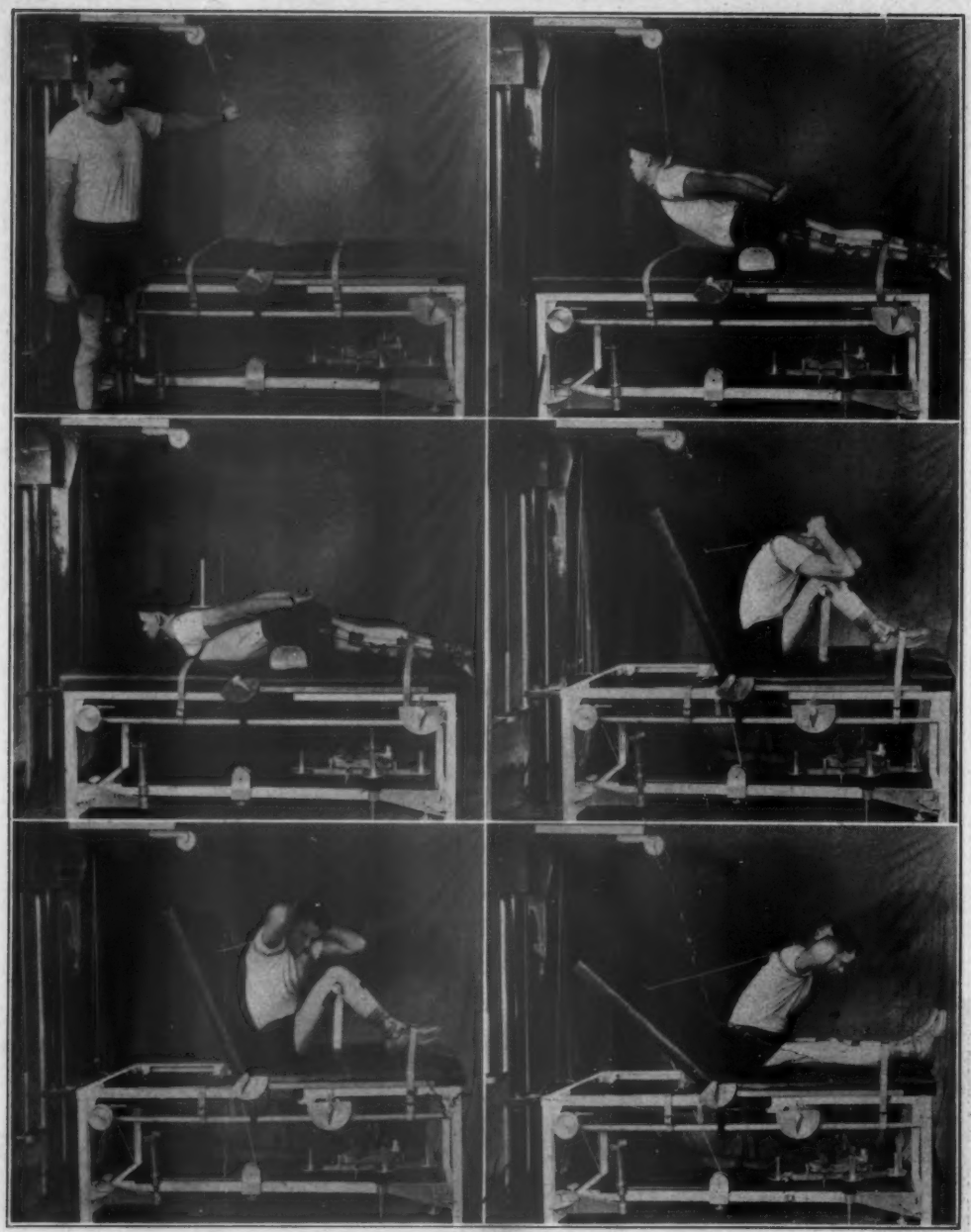

Fig. 13 (top left). — Use of overhead pulley in standing shoulder exercise.

Fig. 14 (top right). — Load-assisting trunk hyperextension.

Fig. 15 (center left). — Load-resisting trunk hyperextension.

Fig. 16 (center right). — Abdominal exercise; hips and knees flexed to eliminate the iliopsoas. For weak abdominals no counterbalancing is needed, as in the sitting position shown; gravity pulls the trunk into flexion. For stronger muscles the exercise load is increased by adding weight to the weight pan, which is attached to the patient's shoulder strap by a rubber cable.

Fig. 17 (bottom left). — Method of stressing oblique components of abdominal muscles.

Fig. 18 (bottom right). — Abdominal exercise; arrangement for bringing hip flexors into action.

strapped to the patient's back (fig. 15). A pillow may be placed under the knees to prevent them from flexing while performing the hyperextension exercise.

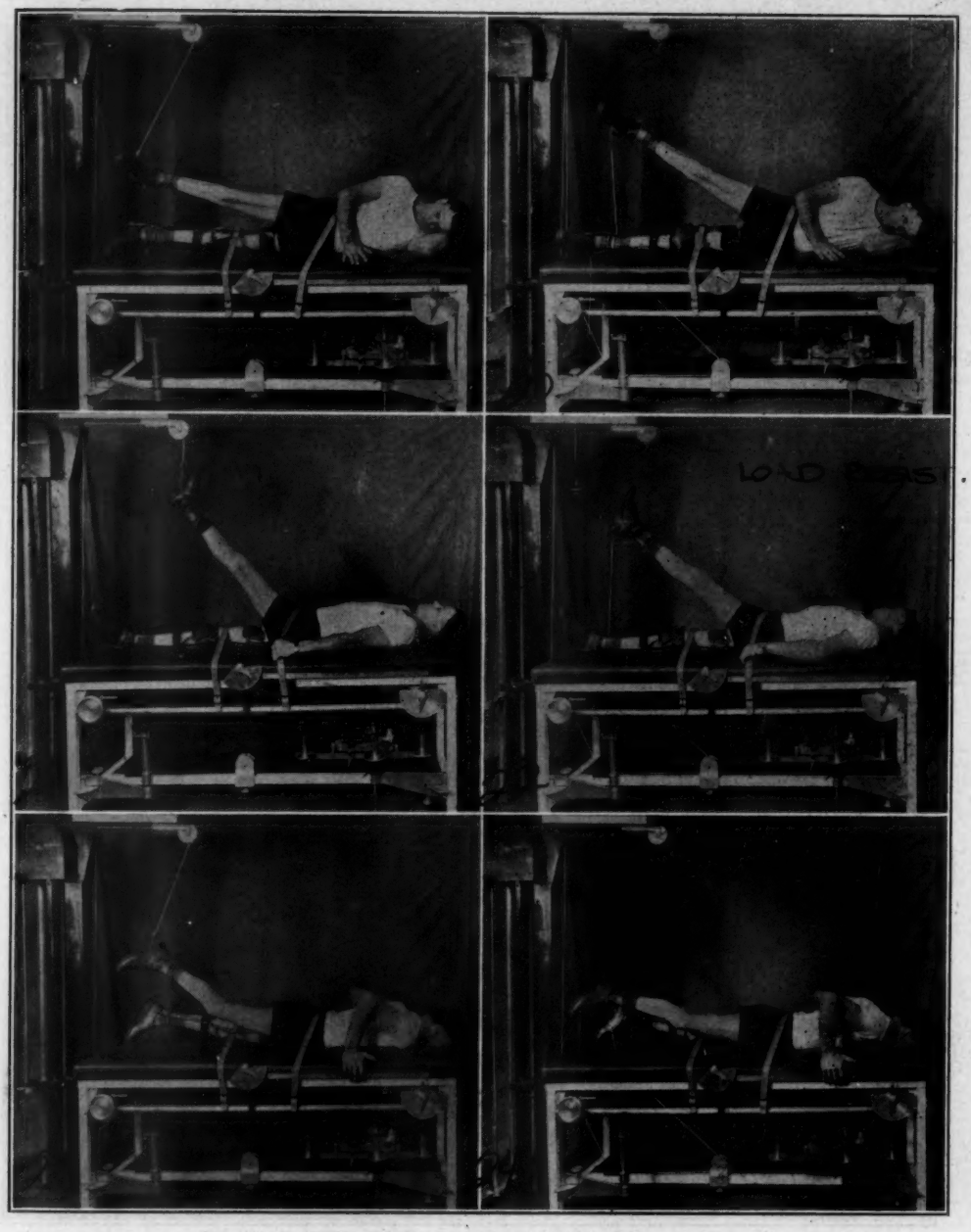

Fig. 19 (top left). — Load-assisting hip abduction exercise.

Fig. 20 (top right). — Load-resisting hip abduction exercise.

Fig. 21 (center left). — Load-assisting hip flexion exercise, or load-resisting hip extension exercise.

Fig. 22 (center right). — Load-resisting hip flexion exercise.

Fig. 23 (bottom left). — Load-assisting hip extension exercise.

Fig. 24 (bottom right). — Load-resisting hip extension exercise.

This exercise also brings the gluteus maximus muscle into action forcefully and may be used for building gluteus maximus power. Experience with this exercise has shown that a weak gluteus maximus muscle does not materially hinder administration of the hyperextension exercise; however, it is difficult to offer resistance to the gluteus maximus in the face of extremely weak spine hyperextensors.

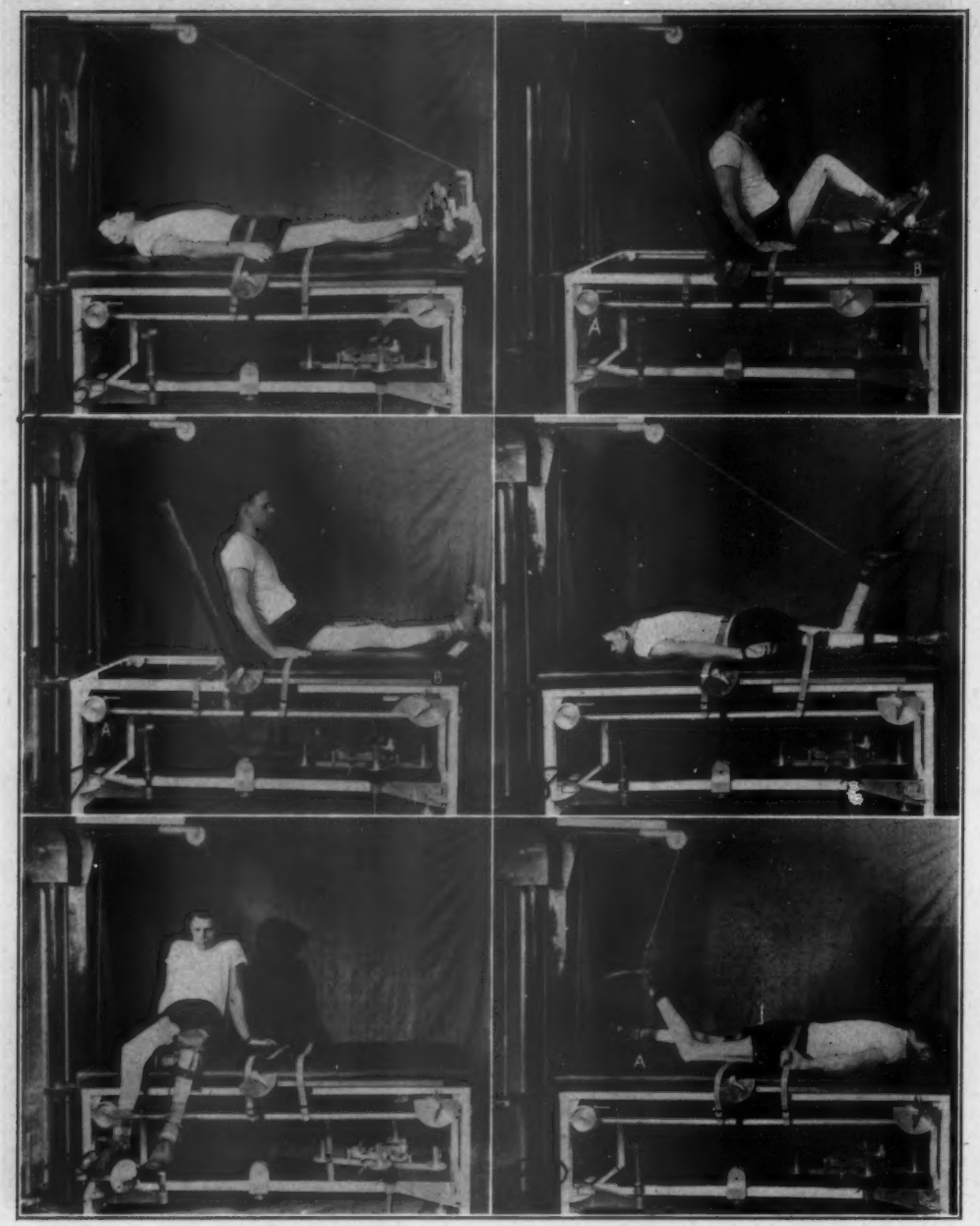

Fig. 25 (top left). — Internal and external hip rotation exercise.

Fig. 26 (top right). — Combined hip and knee extension exercise; first phase. This is an excellent quadriceps femoris and gluteus maximus exercise.

Fig. 27 (center left). — Combined hip and knee extension exercise; second phase. Hip and knee flexion may be stopped at the desired angle by the control knob, *A*. The point to which flexion occurs is indicated on the linear scale, *B*.

Fig. 28 (center right). — Method of exercising a quadriceps femoris muscle that is too weak to complete an arc of motion against gravity.

Fig. 29 (bottom left). — Load-resisting knee extension exercise. Weights are attached to boot which is strapped to the foot.

Fig. 30 (bottom right). — Load-assisting knee flexion exercise. Load-resisting knee flexion may be performed in exactly the same position with resistance offered through the table surface pulley, *A*.

Abdominal Exercises

By maintaining the knees and hips in acute flexion (fig. 16), the iliopsoas action is greatly reduced during trunk flexion and most of the work

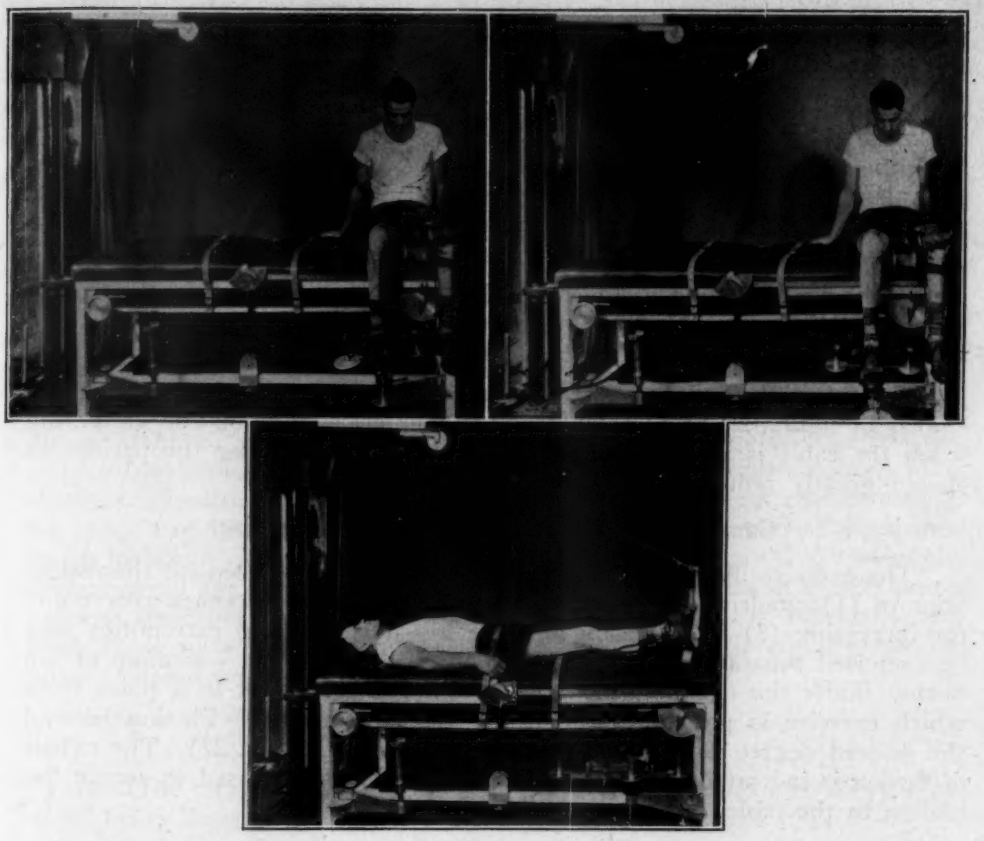

Fig. 31 (left). — Ankle inversion and eversion exercises.

Fig. 32 (right). — Load-resisting dorsiflexion. By adding exercise load to weight pan behind the foot (weight pan not visible), load-assisting dorsiflexion and load-resisting plantar flexion (with flexed knee) are possible.

Fig. 33 (bottom). — Load-resisting plantar flexion with extended knee.

falls on the abdominal muscles. To eliminate the iliopsoas, the hips are flexed until there is no hip motion on trunk flexion. This is a good arrangement when it is desirable to develop the abdominal muscles without concurrent hip flexor development.

By rotating the trunk to the right or left during trunk flexion, the oblique abdominal muscles are stressed (fig. 17).

By performance of the exercises with knees and hips in extension, the iliopsoas is brought into action. This may be accomplished by lowering either the back rest or knee prop or both (fig. 18).

Hip Exercises

The originally described hip exercises were all of the weight-bearing variety. Because of the extreme difficulty in preventing substitution, these were discarded and non-weight-bearing hip exercises developed, which have proved far more effective.

A. Hip Abduction. — By using straps as shown, substitution can be reduced (figs. 19 and 20). If the patient is unable to maintain the knee rigid during these hip exercises because of poor quadriceps muscle power, a light posterior splint may be applied across the knee joint and held in place by an elastic bandage. While performing the abduction exercise, it is advisable to keep the trunk rolled slightly forward to help eliminate tensor fasciae latae substitution.

B. Hip Flexion. — Load-assisting and load-resisting exercises are illustrated in figures 21 and 22.

C. Hip Extension. — Load-assisting and load-resisting hip extension exercises may be performed as illustrated in figures 23 and 24. When they are performed in this manner, it is possible to hyperextend the hip. The exercise may also be performed as shown in figures 21, 26 and 27. However, neither of these exercises allows full extension.

D. Hip Adduction. — The arrangement shown in figure 19 is used for load-assisting abduction. No counterbalancing arrangement is needed for the adduction exercise, for when the limb is abducted no power is required to pull the limb into adduction because of gravity.

E. Internal and External Hip Rotation. — The simple rotator attachment shown in figure 25 provides internal and external rotation for either hip when the cable is wound in the right direction. Strapping the patient as shown greatly reduces substitution.

Combined Hip and Knee Extension Exercise

The exercise illustrated in figures 26 and 27 may be used for the restoration of (1) quadriceps femoris power and (2) gluteus maximus power and for increasing (3) knee flexion and (4) hip flexion. The extremities may be exercised separately or simultaneously. When marked limitation of hip flexion limits the exercise, the back rest may be lowered to a point from which exercise is possible through the available motion. Flexion beyond the desired degree is limited by turning the knob *A* (fig. 27). The extent of flexion is indicated by the linear scale, *B*. Straps are used to secure the patient to the table.

Knee Exercises

A. Knee Extension. — The method of exercising the knee extensors shown in figure 28 makes possible complete extension for a weak quadriceps femoris muscle that cannot complete an arc of motion against gravity. If the quadriceps femoris muscles are of equal power, they may be exercised simultaneously by putting a leather stirrup on each foot and attaching both stirrups to the cable. Straps are useful here for preventing trunk motion of both the exercising and the nonexercising extremity. When there is sufficient quadriceps femoris power to complete an arc of motion against gravity, resistance is added directly to the foot as shown in figure 29. Figures 26 and 27 illustrate another quadriceps femoris exercise, but this exercise does not build power of the vastus medialis component as rapidly or to the extent of the one shown in figure 29.

B. Knee Flexion. — Only the load-assisting knee flexion exercise is illustrated (fig. 30). For a load-resisting, knee flexion exercise a weight on an aluminum boot may be attached to the foot or the cable on the table surface may be attached to leather stirrup. The knee is then flexed as much as to 90 degrees.

Ankle Exercises

A. Inversion and Eversion. — These exercises are illustrated in figure 31. Anterior-posterior motion is eliminated by turning the proper handle. Then either foot may be exercised in inversion or eversion by adding resistance to the appropriate weight pan.

B. Dorsiflexion and Plantar Exercises. — Dorsiflexion is accomplished by blocking out lateral motion and adding weight to the weight pan near

the toes (fig. 32). This is load-resisting dorsiflexion. Load-assisting dorsiflexion is accomplished by adding the exercise load to the weight pan behind the heel. This weight pan is not shown in figure 32.

Plantar flexion may be performed with the knee flexed (fig. 32) or extended (fig. 33). When the exercise is performed in the former manner, the gastrocnemius muscle is relaxed and is not forcefully brought into action, thus throwing most of the work on the plantar flexors. Performed with knee straight, this exercise is excellent for the gastrocnemius. Strapping as shown reduces substitution.

Other Exercises

There are many other possible exercise arrangements that cannot be discussed here. For example, no mention has been made of load-resisting and load-assisting neck exercises (flexion, extension, lateral bending), other shoulder exercises, etc. Most remedial exercise problems encountered in the clinic can be handled efficiently with the versatile exercise equipment which has been used for illustration. It is practically impossible to exercise effectively all age groups, body weights, heights and grades of muscle power without specialized remedial exercise equipment. This is especially true when these numerous variations are associated with different degrees and types of deformity.

Summary

1. The term "progressive resistance exercise" has replaced the original term "heavy resistance exercise" for the reasons mentioned.

2. The principle of counterbalancing has been employed to make possible the administration of progressive resistance exercise to muscles so weak that they cannot complete an arc of motion against gravity.

3. The principles employed in counterbalancing weak muscles are in no way contradictory to those of the original "heavy resistance exercises."

4. The exercise load is the load with which the exercise is performed, either resisting or assisting the muscle.

5. The muscle load is the actual resistance the muscle must overcome.

6. Load-assisting exercises are those in which the exercise load assists the muscle — i. e., counterbalancing.

7. Load-resisting exercises are those in which the exercise load resists the muscle.

8. The increases in muscle loads for the load-resisting exercises are based on the 10 repetition maximum.

9. The increases in muscle loads for the load-assisting exercises are based on the 10 repetition minimum.

10. The 10 repetition maximum is the most weight the muscle can correctly lift 10 times.

11. The 10 repetition minimum is the least amount of help required by the muscle to help it to lift the extremity correctly 10 times through a full arc of motion.

12. Specialized exercise equipment makes it possible to treat patients more efficiently with various complicated remedial exercises.

EFFECTS OF ARTIFICIALLY INDUCED FEVER ON THE CIRCULATION IN ARTHRITIC PATIENTS *

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Artificially induced fever is employed clinically in the treatment of a number of diseases. In chronic arthritis, especially of the rheumatoid type, it seems to be a valuable adjunct. There are several methods that are used for artificial induction of fever. According to the French physiologist Richet,[1] the various types of hyperthermia may be classified into two important groups: (1) passive fever, which is undergone by the organism and (2) active fever, which is aroused by the organism. Hot baths, long wave diathermy, short wave, the Kettering hypertherm (hot humid air), luminous and nonluminous heat cabinet are included among the means of inducing passive fever. Under such conditions the body makes no effort to raise its own temperature. Effort is exerted to become cooler; in other words, the heat-eliminating mechanisms usually are put into action.

Active fever is aroused by the organism and may be induced by injection of foreign proteins, malarial parasites, typhoid vaccine or some colloidal substances. In this type of fever, the organism makes an effort to raise its own temperature and the cells are very active in producing it. The cells have been stimulated either directly or indirectly through the nervous system. This is an endogenous febrile reaction. Accompanying the active fever induced by foreign proteins or by typhoid vaccine administered intravenously, the manifestations of shock usually appear, whereas with the passive fever induced by physical means there are no manifestations of shock.

Chronologically the phases of the febrile reaction produced by intravenous administration of typhoid vaccine can be arranged as follows: (1) prodromal phase, (2) chill phase, (3) flush phase and (4) defervescence phase.

Some of the more important signs and symptoms of these phases are the following: 1. During the prodromal phase yawning, stretching, general malaise, abdominal unrest, nausea, chilliness, pallor and weakness with some headache usually appear. 2. During the chill phase there is a general increase of muscle tone and a definite feeling of cold with shivering, chattering of teeth, severe pallor, cyanosis, low skin temperature and rising body temperature. 3. During the flush phase there is a general feeling of warmth and relaxation with congestion, redness, sweating and high skin and internal temperatures. 4. During the defervescence phase there occurs a gradual diminution of congestion, redness and body temperature.

The following are some of the salient characteristics of hyperthermia produced by physical means: The prodromal phase and its manifestations are

* Read at the Twenty-Fifth Annual Session of the American Congress of Physical Medicine, Minneapolis, Sept. 3, 1947.

1. Richet, Charles, Jr.: Posology of Therapeutic Fever, in Simpson, W. M., and Bierman, William: Fever Therapy; Abstracts and Discussions of Papers Presented at the First International Conference on Fever Therapy, New York, Paul B. Hoeber, Inc., 1937, pp. 18-20.

absent; the chill phase and its manifestations are absent; the flush phase develops gradually from the onset with consistent increase of blood flow, and defervescence usually sets in as soon as the physical agent used as a source of heat is discontinued.

The beneficial effects obtained from the induction of hyperthermia are not necessarily due to the fever per se, for certain treatments have helped without elevation of body temperature. This has led to the use of the expression "pyrotherapy without fever." Furthermore, a patient may have a very high fever and yet get no benefit from it. If it should ever be proved that the benefit derived from pyrotherapy is due to the fever per se, then the hyperthermia produced by physical means would be the method of choice; because, as mentioned previously, this type of fever is associated neither with prodromata nor with chills and the manifestations of shock.

Why does fever occur in the organism? In general, it can be definitely stated that fever occurs as a result of imbalance of the heat-producing and heat-eliminating mechanisms. If heat loss is reduced or if heat production is increased without an accompanying increase of heat elimination, a rise of body temperature is bound to occur. Hardy[2] stated that a normally functioning heat-eliminating mechanism can rid the body of excess heat up to twelve times the basal heat production. If this heat were conserved, it would suffice to raise the temperature of the body to 50 C. (122 F.) in thirty-five minutes. A relatively small increase of heat production, if associated with a large reduction of heat loss, such as occurs in malarial chills, can produce very high fever. This shows that in the development of fever the mechanism of heat loss is as important as that of heat production. Fever can be easily induced by hot baths, by hypertherm cabinets (hot humid air) or by luminous and nonluminous heat cabinets because under such conditions the body is unable to use the skin to eliminate its heat as it normally does. As mentioned previously, the fever produced by such physical agents is neither ushered in by a prodromal phase nor preceded or followed by chills. Furthermore, the flush phase of this type of fever is associated with extreme vasodilatation. In fact, the pink color of the skin all over the body is a prominent feature of fever induced by physical means.

The growing interest in fever therapy makes a careful investigation of the effect of induced fever on the circulation in man especially valuable. Since artificial fever is used extensively in physical medicine in the treatment of various diseases, we wished to study the effects of these types of fever on the circulation in man. Even though the claims for the benefits of artificially induced fever are not attributed solely to the improvement in the circulation and increased blood flow, we considered it instructive and interesting to determine the effects on the circulation of an artificially induced general rise in body temperature. Since so many patients are given fever treatment by means of hot baths or hot humid air (Kettering hypertherms) and also typhoid vaccines when given intravenously, in the treatment of patients who have rheumatoid arthritis, produce high fever, we were interested in comparing the effects on the circulation of these various types of artificially induced fever.

Carefully induced and regulated hyperthermia is useful in more than one way. Ober[3] stated that heat is useful whenever it relieves pain, swelling or spasm or through its influence on the circulation facilitates defensive or repair processes.

2. Hardy, J. D.: Mechanism of Heat Loss from the Human Body, in Simpson, W. M., and Bierman, William: Fever Therapy; Abstracts and Discussions of Papers Presented at the First International Conference on Fever Therapy, New York, Paul B. Hoeber, Inc., 1937, pp. 16-17.
3. Ober, F. R.: Heat in Surgical and Orthopedic Conditions, J. A. M. A. 126:769 (Nov. 18) 1944.

Stecher and Solomon[4] stated that the mechanism producing benefit from artificial fever by means of the Kettering hypertherm in arthritis is not clear, but definite physiologic effects are produced—namely, a substantial rise of leukocytes and an increase of blood flow, of the cardiac output, of metabolism and of the number and size of capillaries.

Ghormley and Silverglade[5] found that general heating of the body is more efficient than the local application of heat to the diseased joint. Hewlett[6] and Abramson and associates[7] reported that application of heat to the hands or other peripheral regions of the body led to dilatation of the cutaneous vessels and to a twofold or threefold increase of the rate of blood flow. The oxygen saturation of venous blood approached that of arterial blood when the arm was exposed to baths at temperatures of 40 to 46 C.[8] Grollman[9] reported an increase of cardiac output when the environmental temperature was raised beyond 30 C. This increase of cardiac output and the increase of heart rate help to maintain the blood pressure in spite of the vasodilatation and reduced peripheral resistance. The contraction of the spleen under the influence of heat contributes to the maintenance of adequate venous return by increasing the blood volume. Barcroft and Edholm[10] produced a rise of body temperature and increased blood flow in the forearm by immersing the feet and ankles in water at 44 to 45 C. while the test arm was kept in a water bath at 30, 32 and 35 C. They suggested a water bath temperature between 33 and 35 C. as the most satisfactory. Pinkston[11] stated that general peripheral vasodilation occurs almost immediately when external heat is applied, but when the fever is induced by foreign protein a period of peripheral vasoconstriction with lowering of cutaneous temperature precedes the vasodilation and the period of dissipation of heat. This idea is also held by Kissin and Bierman.[12] In the foot at room temperature, Killian and Oclassen[13] obtained blood flow of 0.9 to 4.2 cc. per minute per 100 cc. of limb volume, but after application of local heat or mustard the flows were 6.4 to 14.8 and 7.3 to 13.2 cc. per minute per 100 cc. of limb volume, respectively. Kunkel and Stead[14] obtained maximal flows at 43 C. which were 11.1 to 25.9 cc. per minute per 100 cc. of limb volume.

Prusík[15] stated that the vascular changes in the skin of patients who had rheumatoid arthritis consisted in venous congestion in the skin over the inflamed joints. Woodmansey and associates[16] observed that patients who had rheumatoid arthritis responded poorly to contrast baths as compared with

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4. Stecher, R. M., and Solomon, W. M.: The Treatment of Acute Nonspecific Infectious Arthritis with Artificial Fever, in Simpson, W. M., and Bierman, William: Fever Therapy; Abstracts and Discussions of Papers Presented at the First International Conference on Fever Therapy, New York, Paul B. Hoeber, Inc., 1937, pp. 116-117.

5. Ghormley, J. W., and Silverglade, Alexander: Circulation of the Joints of Chronic Arthritis, New York State J. Med. 39:1489 (Aug. 1) 1939.

6. Hewlett, A. W.: The Effect of Room Temperature Upon the Blood Flow in the Arm, with a Few Observations on the Effect of Fever, Heart 2:230, 1910.

7. Abramson, D. I.; Zazeela, Herman, and Marrus, Joseph: Plethysmographic Studies of Peripheral Blood Flow in Man. II. Physiologic Factors Affecting Resting Blood Flow in the Extremities, Am. Heart J. 17:206 (Feb.) 1939.

8. Goldschmidt, Samuel, and Light, A. B.: The Effect of Local Temperature Upon the Peripheral Circulation and Metabolism of Tissues as Revealed by the Gaseous Content of Venous Blood, Am. J. Physiol. 73:146 (June 1) 1925.

9. Grollman, Arthur: Physiological Variations of the Cardiac Output in Man: X. The Effect of Variations in the Environmental Temperature on the Pulse Rate, Blood Pressure, Oxygen Consumption, Arteriovenous Oxygen Difference, and Cardiac Output of Normal Individuals, Am. J. Physiol. 95:263 (Nov. 1) 1930.

10. Barcroft, H., and Edholm, O. G.: Temperature and Blood Flow in the Human Forearm, J. Physiol. 104:366 (Apr. 15) 1946.

11. Pinkston, J. O.: Peripheral Circulation During Experimental Fever, Am. J. Physiol. 110:448 (Dec.) 1934.

12. Kissin, Milton, and Bierman, William: Influence of Hyperpyrexia on Velocity of Blood Flow, Proc. Soc. Exper. Biol. & Med. 30:527 (Jan.) 1933.

13. Killian, J. A., and Oclassen, C. A.: Comparative Effects of Water Baths and Mustard Baths at Varying Temperatures on the Rate of Peripheral Blood Flow in Man, Am. Heart J. 15:425 (Apr.) 1938.

14. Kunkel, P., and Stead, E. A., Jr.: Blood Flow and Vasomotor Reactions in Foot in Health, in Arteriosclerosis, and in Thromboangiitis Obliterans, J. Clin. Investigation 17:715 (Nov.) 1938.

15. Prusík, Bohumil: Blood Circulation of the Skin in Rheumatism, Acta Rheumatol. 2:17 (Nov.) 1930.

16. Woodmansey, A.; Collins, D. H., and Ernst, M. M.: Vascular Reactions to the Contrast Bath in Health and in Rheumatoid Arthritis, Lancet 2:1350 (Dec. 10) 1938.

normal persons. They concluded that the attenuated blood supply resulted from disuse of the parts involved rather than from any vasospastic condition. Steinbrocker and Samuels[17] concluded that vasospastic disturbances were common among patients who had rheumatoid arthritis. Rowntree, Adson and Hench[18] found that after removal of the lumbar sympathetic trunks and ganglions they obtained an increase of 5 to 10 C. in the cutaneous temperature of the fingers and toes in patients who had chronic arthritis. They suggested that after sympathetic resection the arteriolar spasm ceases and more rapid blood flow occurs. Wright and Pemberton[19] reported that most patients who had arthritis showed a reduction of surface temperature at the base of the finger nails. They also noted that the capillaries contained less blood and were paler than those of normal persons. They suggested that either fewer capillaries were present or the individual vessels were smaller. Other authors,[20] reported reduced blood flow in arthritis.

Altschule and Freedberg[21] stated that during the prodromal phase of an endogenous febrile reaction no significant physiologic or chemical alterations can be observed but the chill phase is characterized by a sympathetic discharge leading to marked generalized vasoconstriction which causes circulatory stasis of variable degrees.

Johnson and associates[22] reported that the spasm of cutaneous blood vessels during the chill was due to activity of the sympathetic system and that in sympathectomized limbs there was no spasm of the cutaneous vessels during a chill caused by injection of typhoid vaccine.

Methods

In the study of the blood flow in man the methods to be employed must involve neither excision nor ligation of vessels nor exposure of the blood and tissues to any dangers of infection. In other words, the methods must be indirect and the body should be kept intact. To date two methods have been used for the study of blood flow in man: (1) the plethysmographic method, and (2) the calorimetric method. Hewlett and van Zwaluwenburg[23] were the originators of the venous occlusion plethysmographic method for quantitative study of the circulation in the extremities of man.

In 1912 Stewart[24] developed a different type of quantitative procedure for the study of blood flow in human limbs. This method determines the rate of blood flow from the rate of heat loss from the limb. Abramson[25] stated that this method is not suitable for measuring sudden changes of blood flow and, furthermore, that the readings are an index of cutaneous blood flow rather than of total flow to an extremity.

In general, such procedures, calorimetric or plethysmographic, furnish information regarding the responses of the vascular tree and some of the

17. Steinbrocker, Otto, and Samuels, S. S.: The Arterial Circulation of the Lower Extremities in Chronic Arthritis, J. Lab. & Clin. Med. 26:974 (Mar.) 1941.
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20. Weil, A. J.: Zur Frage des Anteils der Gefässe bei arthritischen Erkrankungen, Acta Rheumatoil. 2:47 (Nov.) 1930. Kovács, Joseph; Wright, Irving, and Duryee, A. W.: The Surface Temperature and Minute Blood Vessels of the Skin in Arthritis, J. A. M. A. 100:1018 (Apr. 1) 1933. Prusik.15.
21. Altschule, M. D., and Freedberg, A. S.: Circulation and Respiration in Fever, Medicine 24:403 (Dec.) 1945.
22. Johnson, C. A.; Osborne, Stafford, and Scupham, George: Studies of Peripheral Vascular Phenomena: IV. The Effect of Artificial Fever on the Pulse Volume Change of the Finger, Am. J. M. Sc. 190:485 (Oct.) 1935.
23. Hewlett, A. W., and van Zwaluwenburg, J. G.: The Rate of Blood Flow in the Arm, Heart 1:87, 1909.
24. Stewart, G. N.: Studies on the Circulation in Man. II. The Effect of Reflex Vasomotor Excitation on the Blood Flow in the Hand, Heart 3:76, 1911-1912.
25. Abramson, D. I.: Vascular Responses in the Extremities of Man in Health and Disease, Chicago, University of Chicago Press, 1944, p. 53.

effects of the autonomic nervous system on the circulation. The calorimetric method does not have as many supporters as the plethysmographic method because it is believed to have many disadvantages.

The plethysmographic method used in this study has been described in detail elsewhere[26]; therefore, only a few of the main features will be given in this communication. The apparatus consists of two plethysmographs — the true plethysmograph and the blank — placed one on each of the upper extremities to cover the whole forearm and about 1 inch (2.5 cm.) above the olecranon process. Each plethysmograph is connected to a spirometer for volume recording. When both plethysmographs and spirometers are all functioning as a unit, a nicely compensating mechanism eliminates errors produced by changes in room temperature and pressure. The compensating mechanism is described in detail in another communication.[26] The plethysmograph is made airtight over the limb without any interference with the circulation. This is done by placing a thin sleeve of rubber dam over the arm, which is coated with surgical jelly above the elbow. The sleeve is everted over a diaphragm of sponge rubber. The limb is introduced into the plethysmograph and the diaphragm is clamped by a metal ring to the border of the plethysmograph, thus making an airtight compartment over the forearm 1 inch (2.5 cm.) above the elbow and communicating with the spirometers by means of a wide bore tubing. A blood pressure cuff, 5 cm. wide and connected to a reservoir of air under a pressure of 50 to 60 mm. of mercury, is wrapped around the part of the arm proximal to the true plethysmograph. When this cuff is suddenly inflated to a pressure of about 50 mm. of mercury, well below diastolic but above venous pressure, it traps the blood in the limb until the venous pressure exceeds the pressure in the cuff. For several seconds after the "collecting cuff" is suddenly inflated, the volume of the limb increases because of hindrance of venous outflow without interference with arterial inflow into the limb.

In principle, the amount of blood flowing into the extremity is measured by determining the rate of increase of volume subsequent to the obstruction to venous outflow in the limb. The increase of volume of the limb, which occurs for several seconds after sudden inflation of the collecting cuff, indicates the rate of blood flow into the limb. When the rising venous pressures exceed the pressure in the collecting cuff, blood begins to escape through the veins, and subsequently the increasing volume starts to decline and cause plateauing of the flow curve. Increased resistance in the engorged limb impedes arterial inflow and contributes to the decline and plateauing of the curve. There is usually a collecting cuff artefact, the magnitude of which varies usually with the pressure in the collecting cuff and with the distance between the cuff and the plethysmograph. The artefact is primarily produced by displacement of the fluid and tissues under the cuff. We have come to realize that it is practically impossible to eliminate the cuff artefact completely unless the plethysmograph is left open during inflation of the cuff. This maneuver, of course, introduces other and more serious errors. Therefore, we have found it more advisable to apply the collecting cuff at a fixed distance from the plethysmograph. Since the part of the curve produced by the artefact is exceedingly steep and shows no pulsation, it can be easily ignored and the blood flow is computed from the rest of the flow curve.

Shape of the Flow Curve. — In general, it can be stated that the height at which a flow curve begins to plateau indicates the capacity of the vascular tree of the limb, as a whole, and the small vessel bed, in particular, distal to the cuff.

It was repeatedly observed in this study that the control flow curves, the values of which were much lower than those after induction of fever, consistently showed plateauing at low levels. Those with exceedingly high flows swept beyond the 12 inch paper in the camera before plateauing. This suggested a vast vasodilatation in the vascular tree and a great increase in its capacity and in blood flow after induction of fever.

26. Berry, M. R.; Baldes, E. J.; Essex, H. E., and Wakim, K. G.: A Compensating Plethysmokymograph for Measuring Blood Flow in Human Extremities, J. Lab. & Clin. Med. 33:101 (Jan.) 1948.

Two factors contribute to the plateauing and to the decreasing increments of volume produced by successive beats which form the flow curve: (1) a progressive decrease of blood flow due to the interference by venous congestion and (2) the escape of venous blood past the collecting cuff after the venous pressure distal to the inflated cuff rises above the pressure within the cuff. In fast flows the curve and the venous pressure rise so quickly that only the first three seconds can be used for computing blood flow from the curve, while in very slow flows five seconds or more may be utilized.

Adult patients who were being treated for arthritis were used in this study during the period when they were scheduled for various types of fever treatment — Kettering hypertherm (cabinet fever), hot tub baths or typhoid vaccine administered intravenously. The blood flow in the left forearm of each subject was determined by means of the compensating plethysmokymograph as described previously.[26] No attempts were made to establish basal values, especially because the observations had to be made at any time during the day when the patient was scheduled for fever treatment. In every case several control determinations of blood flow were made immediately before the patient was sent for induction of fever. The subject reclined comfortably on the test bed and after control oral temperature, blood pressure and heart rate had been determined the upper extremities were placed in the plethysmographs to a level approximately 1 inch (2.5 cm.) proximal to the olecranon process. During a period of about fifteen minutes several control blood flow determinations were made. Soon after induction of hyperthermia the patient was brought back to the observation room, and the blood pressure, oral temperature and heart rate were again recorded. Within about ten minutes after induction of fever to about 101 F., the patient was prepared as described and blood flow determinations were again made from the left forearm once every two and a half minutes for a period of forty minutes.[27]

Results

In tables 1 and 2 is presented a summary of the data on 15 patients in whom a temperature of 101 F. was induced by the use of tub baths at temperatures several degrees above that of the body for a period of about half an hour. The control oral temperature ranged from 97.0 to 99.0 F., and ten minutes after the patients had finished the tub bath, when blood flow determinations were started, the range of oral temperature was from 98.2 to 100.3 F. The control blood flow values before induction of fever in these 15 subjects ranged from 23 to 151 cc. per minute per forearm or from 1.8 to 12.5 cc. per minute per 100 cc. of forearm volume. The average control flow was 71 cc. per minute per forearm or 5.2 cc. per minute per 100 cc. of forearm volume. All 15 subjects showed definite increase of blood flow as a result of the fever induced by hot baths. The percentage increase of flow after fever over the control flow in the forearm (before induction of fever) ranged from +22 up to +440 per cent, with an average increase of +170 per cent. Ten minutes after the induction of fever the blood flow in the forearm averaged 11.7 cc. per minute per 100 cc. of forearm volume, as compared with the average control of 5.2 cc.; twenty-five minutes after the induction of fever the flow averaged 10.5 cc. per minute per 100 cc. of limb volume, and after forty minutes it still averaged 9.5 cc. per minute per 100 cc. of limb volume. After long periods of observation during which blood flow determinations were repeatedly made, we noted that it took several hours before the blood flow gradually returned to normal value.

The heart rate increased in every case as a result of the induced fever. The degree of cardiac acceleration varied with the individual and with the temperature. The average increase in these 15 subjects was 10.5 beats for each Fahrenheit degree of rise of temperature. The effects on blood pressure were somewhat variable. The systolic blood pressure showed some rise in

27. We wish to express our appreciation to Mr. Adrien N. Porter for his excellent technical assistance in carrying out this study.

4 subjects, slight fall in 6 and no change in 5. In 11 subjects there was some fall of the diastolic pressure; in 2 there was no change, and in the remaining 2 there was a slight rise. Table 1 represents these data in detail.

The data on the effects of fever induced by the Kettering hypertherm (hot humid air) are presented in tables 3 and 4. The control blood flow, in the

TABLE 1. —

Effects of Artificially Induced Fever (Tub Bath) on the Peripheral Circulation in Man.

			Blood Flow, cc. Per Minute				Heart Rate			Blood Pressure, mm. of mercury		
Subject	Control Temp., F.	Maximal Temp., F.	Before Fever	After Fever	Difference, cc.	Difference, Per Cent	Before Fever	After Fever	Increase	Before Fever	After Fever	Difference
1	98.6	99.3	23	103	80	+347	88	110	22	95/75	110/65	+15/—10
2	98.6	99.8	45	243	198	+440	80	104	24	110/70	110/55	0/—15
3	97.0	99.2	28	99	71	+253	88	128	40	100/80	110/50	+10/—30
4	98.6	99.4	105	191	86	+ 82	88	116	28	120/70	120/70	0/0
5	97.0	99.6	48	98	50	+104	96	106	10	100/60	90/50	—10/—10
6	98.6	99.9	88	195	107	+122	88	118	30	120/70	120/60	0/—10
7	98.6	100.3	69	142	73	+106	88	116	28	110/85	110/80	0/—5
8	97.8	99.5	52	171	119	+229	90	122	32	115/75	120/75	+5/0
9	98.6	99.5	83	115	32	+ 38	98	100	2	120/75	105/60	—15/—15
10	99.0	99.5	68	83	15	+ 22	98	120	22	105/70	100/80	—5/+10
11	98.2	99.5	33	142	109	+330	84	104	20	110/75	110/80	0/+5
12	98.0	99.0	49	160	111	+227	104	132	28	115/80	110/65	—5/—15
13	97.3	99.7	151	276	125	+ 83	88	126	38	110/70	120/60	+10/—10
14	98.9	99.8	85	184	99	+116	95	128	33	130/70	125/65	—5/—5
15	98.8	99.8	143	215	72	+ 50	84	140	56	165/100	150/90	—15/—10
		Average	71	161	90	+170						

forearm before cabinet fever was induced, ranged from 22 to 106 cc. per minute or 1.7 to 8.9 cc. per minute per 100 cc. of forearm volume, with a mean control blood flow before fever of 50 cc. per minute per forearm volume

TABLE 2. —

Effects of Artificially Induced Fever (Tub Bath) on the Peripheral Circulation in Man.

		Temperature After Fever, F.			Blood Flow, cc. Per Min. Per 100 cc.				
Subject	Control Temp., F.	10 Min.	25 Min.	40 Min.	Before	10 Min.	25 Min.	40 Min.	Arm Volume, cc.
1	98.6	99.3	99.3	99.0	2.1	9.3	8.0	6.7	1,100
2	98.6	99.8	99.3	99.3	3.5	18.6	14.8	15.5	1,300
3	97.0	98.2	99.0	98.6	2.8	9.9	8.7	8.1	1,000
4	98.6	99.4	99.2	99.2	5.0	9.1	8.6	8.9	2,100
5	97.0	99.6	99.3	98.9	3.8	7.8	6.6	5.9	1,250
6	98.6	99.9	99.5	99.5	5.9	13.0	12.4	11.4	1,500
7	98.6	100.3	100.0	99.9	5.3	10.9	9.9	10.8	1,300
8	97.8	99.5	98.4	98.4	3.5	11.4	10.1	8.7	1,500
9	98.6	99.5	99.0	98.5	6.4	8.9	8.2	6.8	1,300
10	99.0	99.5	99.3	99.3	8.0	9.5	9.8	9.4	850
11	98.2	99.5	99.2	99.0	1.8	7.3	7.7	5.9	1,850
12	98.0	99.0	99.0	98.8	3.5	11.4	11.3	10.8	1,400
13	97.3	99.7	99.3	99.1	12.5	23.0	18.5	17.0	1,200
14	98.9	99.8	99.3	99.2	5.7	12.2	10.5	7.0	1,500
15	98.8	99.8	99.5	99.4	8.4	12.6	11.7	9.6	1,700
				Average	5.2	11.7	10.5	9.5	.....

or 3.7 cc. per minute per 100 cc. of limb volume. After induction of fever the flow ranged from 75 to 400 cc. per minute, with a mean flow of 163 cc. per minute per forearm volume — an average increase of 113 cc., or +263 per cent. The mean blood flow per minute per 100 cc. of forearm substance was

3.7 cc. before fever and 11.8, 11.5, and 11.0 cc., ten minutes, twenty-five minutes and forty minutes after fever, respectively.

The changes of blood pressure as a result of fever induced by the Kettering hypertherm were as follows: Of the 15 subjects studied, 5 showed some rise in systolic blood pressure, 3 showed no change and the remaining

TABLE 3. —

Effects of Artificially Induced Fever (Cabinet) on the Peripheral Circulation in Man.

			Blood Flow, cc. Per Minute				Heart Rate			Blood Pressure, mm. of mercury		
Subject	Control Temp., F.	Maximal Temp., F.	Before Fever	After Fever	Difference, cc.	Difference, Per Cent	Before Fever	After Fever	Increase	Before Fever	After Fever	Difference
12	98.2	100.5	60	172	112	+187	86	128	42	120/80	100/70	—20/—10
2	98.0	100.2	32	117	85	+265	80	112	32	110/75	115/60	+5/—15
4	98.8	99.9	55	98	43	+ 78	92	96	4	110/70	110/70	0/0
8	98.6	100.2	34	140	106	+312	80	126	46	120/70	115/60	—5/—10
7	98.0	100.4	73	130	57	+ 78	96	122	26	105/80	95/55	—10/—25
16	97.0	100.0	22	87	65	+295	88	164	76	110/75	105/55	—5/—20
17	97.0	100.2	25	165	140	+560	114	146	32	110/65	120/40	+10/—25
15	98.8	100.5	32	160	128	+400	102	126	24	125/80	115/55	—10/—25
3	97.4	99.7	25	75	50	+200	72	120	48	95/70	110/55	+15/—15
11	97.8	99.2	40	112	72	+180	80	120	40	110/75	110/70	0/—5
18	98.0	100.5	35	222	187	+535	80	100	20	120/70	120/40	0/—30
6	96.2	100.8	79	225	146	+185	80	134	54	110/70	130/60	+20/—10
5	97.0	99.0	35	122	87	+249	70	118	48	110/60	100/45	—10/—15
14	99.0	100.0	89	215	126	+142	92	112	20	110/65	100/60	—10/—5
13	97.3	101.5	106	400	294	+277	88	128	40	100/60	110/45	+10/—15
		Average	50	163	113	+263						

7 showed a slight fall. In all subjects there was a fall of diastolic blood pressure, with the exception of 1, in whom no change in diastolic pressure occurred. Table 3 shows these data in detail. The heart rate increased in

TABLE 4. —

Effects of Artificially Induced Fever (Cabinet) on the Peripheral Circulation in Man.

		Temperature After Fever, F.			Blood Flow, cc. Per Min. Per 100 cc.				
Subject	Control Temp., F.	10 Min.	25 Min.	40 Min.	Before	10 Min.	25 Min.	40 Min.	Arm Volume, cc.
12	98.2	100.5	99.7	99.2	4.5	12.3	9.8	7.7	1,400
2	98.0	100.2	99.5	99.1	2.5	7.1	9.0	6.2	1,300
4	98.8	99.9	99.9	99.6	4.2	7.5	7.0	....	1,300
8	98.6	100.2	99.6	99.2	2.3	9.3	9.2	8.3	1,500
7	98.0	100.4	99.9	99.3	5.6	10.0	9.7	9.8	1,300
16	97.0	100.0	99.5	99.0	2.2	7.0	8.4	8.7	1,000
17	97.0	100.2	100.0	100.0	1.7	10.5	9.9	11.8	1,450
15	98.8	100.5	100.5	100.5	3.0	15.1	12.5	10.9	1,050
3	97.4	99.7	99.5	99.1	2.5	7.5	6.7	6.6	1,000
11	97.8	99.2	99.0	99.0	2.2	6.1	5.9	6.3	1,850
18	98.0	100.5	100.1	99.9	2.3	14.3	13.0	12.7	1,550
6	96.2	100.8	100.5	100.1	5.3	13.5	15.0	13.9	1,500
5	97.0	99.0	98.8	98.7	2.8	9.5	9.8	5.9	1,250
14	99.0	100.0	99.5	99.5	5.9	14.3	14.1	11.4	1,500
13	97.3	101.5	100.9	100.8	8.9	33.3	31.9	33.1	1,200
				Average	3.7	11.8	11.5	11.0	.....

every case, with a slight variation in individual cases. The average increase in heart rate was 10.5 beats for each degree (F.) rise in temperature.

Fifteen healthy subjects were used as controls for determination of normal blood flow. Again, no attempts were made to establish basal conditions.

The blood flow in the forearm ranged from 58 to 161 cc., with an average of 92 cc. per forearm or 6.0 cc. per minute per 100 cc. of forearm volume. Table 5 gives the data in detail on each of the 15 persons.

The constitutional reaction after intravenous administration of typhoid vaccine appeared from half an hour to several hours after the injection. As

TABLE 5. — *Blood Flow on Normal Controls (Left Forearm).*

Subject	Blood Flow, cc. Per Min. Total	Blood Flow, cc. Per Min. Per 100 cc. Tissue	Arm Volume, cc.	Body Temperature, F.
19	74	8.2	900	97.5
20	81	6.1	1,450	98.0
21	132	6.3	2,100	98.4
22	93	7.7	1,200	98.3
23	158	7.9	2,000	------
24	66	5.5	1,200	97.8
25	80	4.3	1,850	97.8
26	72	4.2	1,700	98.4
27	96	4.5	2,100	97.6
28	161	10.4	1,550	97.0
29	72	4.0	1,800	97.3
30	58	4.2	1,350	------
31	91	7.0	1,300	98.6
32	81	5.1	1,600	98.4
33	64	4.6	1,400	97.8
Average flow	92	6.0	--------	------

TABLE 6. — *Effect of Intravenously Administered Typhoid Vaccine on the Blood Flow.*

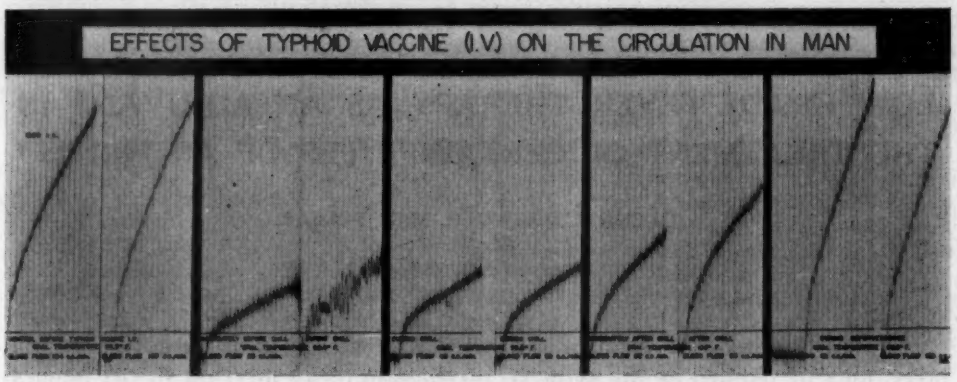
Subject	Control Flow, cc. Per Min.	During Chill Phase cc. Per Min.	During Chill Phase Per Cent Change	During Flush Phase cc. Per Min.	During Flush Phase Per Cent Change
34	70	46	—34	127	+82
35	121	46	—62	161	+38
36	105	32	—69	167	+59
37	152	81	—47	188	+24
38	112	40	—64	------	--------
39	78	40	—49	------	--------
40	80	----	--------	126	+58
41	57	----	--------	65	+14
42	103	92	—12	------	--------
43	58	----	--------	81	+40

stated previously, the reaction started with malaise and aches throughout the body, and in many cases definite and severe shaking chills took place. These reactions were soon followed by a general feeling of warmth with definite fever, the temperature being 100 F. or more. After a few hours of fever and sweating, the temperature gradually returned to the preinjection level. The changes in the circulation were as follows: During the prodromal phase there was a slight cutaneous pallor with reduction of blood flow. This reduction of blood flow became maximal at the peak of the chill phase, during which severe pallor and goose flesh were marked. Soon after the chill a general flushing of the skin occurred and the blood flow increased above the control level. This lasted throughout the period of defervescence, toward the end of which the blood flow gradually returned toward the control level. Before and at intervals after intravenous injection of typhoid vaccine, the blood flow was determined in 10 adult patients who had rheumatoid arthritis involving various joints. Control blood flow determinations were made immediately before the injection of typhoid vaccine. Similar blood flow determinations were also made before the chill, during the chill, at the end of the chill, during the flush and during the defervescence phases after various doses of typhoid vaccine had been administered intravenously. In table 6 the data on these 10 patients are presented. The consistent reduction of

TABLE 7. — *Comparative Effects of Fever Induced by Intravenous Typhoid Vaccine, Kettering Hypertherm (Cabinet), and Hot Tub Baths on the Blood Flow in the Forearm of Arthritic Patients.*

Subject	Percentage Deviation in Blood Flow from the Control			
	Intravenous Typhoid Vaccine		Kettering Hypertherm	Tub Bath
	Chill Phase	Flush Phase	Fever	Fever
M. C.	—34	+82	+185	+122
J. B.	—62	+38	+277	+ 83
C. H.	—69	+59	+250	+294
M. W.	—47	+24	+194	+107
Average	—53	+51	+226	+152

blood flow, which was noted for some time before the chill, became maximal during the chill. During the flush phase there was an increase of blood flow, but the magnitude of this increase was less than that produced by hot tub baths or cabinet fever producing a corresponding rise in body temperature.



Blood flow curves taken before and during the various phases after intravenous injection of typhoid vaccine. Note the reduction of blood flow before and during the chill and the moderate increase during defervescence.

The figure demonstrates the changes of blood flow as seen in the flow curves taken during the various stages after typhoid vaccine injections. Table 7 gives a comparison of the changes of blood flow produced by the various agents used in the induction of artificial fever. Note the consistent decrease of blood flow during the chill produced by typhoid vaccine injected intravenously.

Summary

By the use of the compensating spirometer recorder the blood flow was studied plethysmographically, before and after artificial induction of fever, in patients who had rheumatoid arthritis involving various joints. For therapeutic purposes artificial fever was induced actively by the intravenous administration of typhoid vaccine and passively by physical means — Kettering hypertherm and hot tub baths — to a temperature of about 101 F. or more. Induced hyperthermia, whether active or passive, produced an increase of the peripheral circulation. Active fever induced by intravenous administration of typhoid vaccine led to a reduction of peripheral blood flow during the chill phase. Physically induced fever, by the use of hot baths or Kettering hypertherm, was consistently accompanied by an increase of blood flow and an increase of heart rate. Prodromal symptoms and chills characterized the fever artificially induced by the intravenous administration of typhoid vaccine but were nonexistent in the physically induced fever.

Discussion

Dr. W. A. Selle (Galveston, Texas: I am sure that all of us who have followed the literature on blood flow in hyperthermia welcome the contribution of Dr. Wakim and his colleagues. This is a matter of practical importance to the physiatrist and of much academic interest to the physiologist. Fundamental studies such as this will help us to establish a more rational basis for the practice of physical medicine.

I should like to ask Dr. Wakim a question. Were you able to determine whether or not the untoward effects of hyperthermia, sometimes observed during the induction stage, are due to inadequate filling of the heart which might result from the high rate of contraction and reduced diastasis?

Dr. Wakim (closing): To answer Dr. Selle's question, I should like to say that the temperatures of our patients were not raised above 102 F. I tried to stress the idea that it is not necessary to raise the patients' temperatures too much; 101 and 102 is an exceedingly good temperature. There are certain diseases, however, in which you have to raise the temperatures to much higher levels. Perhaps patients with those diseases are the ones that Dr. Selle has in mind; their heart rates may go to speeds at which circulatory disturbances could occur.

LOW GRADE FEVER THERAPY AS AN ADJUVANT IN THE TREATMENT OF CERTAIN TYPES OF ARTHRITIS *

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Fever therapy, the term now applied to systemic heating of the human body, may be defined as the artificial production of an increase in the general bodily temperature for therapeutic purposes.[1] It produces certain physiologic changes in the human patient: 1. There is an increase in the leukocyte count[2] and a decrease in blood platelets.[3] 2. The *p*H of the blood is increased, the carbon dioxide-combining power decreased and the blood temperature increased.[4] 3. There is little or no change in the sedimentation rate[5] or specific gravity of the blood.[6] 4. There is increased ketosis, with an increased rate of ketogenesis in the liver.[7] 5. The electrolytes of the blood show little, if any, alteration.[8] 6. Studies of the blood ascorbic acid level reveal no significant change.[9] 7. There is an initial rise in systolic blood pressure and a slight drop in the diastolic pressure; subsequently there is a tendency toward a decrease in both the systolic and the diastolic pressure. 8. The velocity of blood flow is increased, with a consequent decrease in circulation time.

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* From the Department of Medicine, Glenwood Hot Springs Clinic.

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2. Krusen, F. H.: The Blood Picture Before and After Fever Therapy by Physical Means, Am. J. M. Sc. 193:470 (April) 1937. Hargraves, M. M., and Doan, C. A.: The Physiologic Response of the Hemopoietic Tissues to Artificially Induced Fever, in: Abstracts of Papers and Discussions, Fifth Annual Fever Conference, May 2 and 3, 1935, pp. 51-58.
3. Wilson, S. J., and Doan, C. A.: Pathogenesis of Hemorrhage in Induced Fever, Ann. Int. Med. 13:1214, (Jan.) 1940.
4. Ferguson, C., and Buckholtz, M.: Effect of Hyperpyrexia on pH of Blood, Arch. Phys. Therapy 22:333 (June) 1941. Osborne, S. L.; Markson, D. E., and Merriman, J. R.: J. Lab. & Clin. Med. 27:1135 (June) 1942.
5. Fetter, F., and Schnabel, T. G.: Behavior of Blood Sedimentation Rate During and After Fever, Am. J. M. Sc. 201:115 (Jan.) 1941.
6. Osborne, S. L.: Hyperpyrexia and Specific Gravity of Blood, Arch. Phys. Therapy 22:407 (July) 1941.
7. Somogyi, M., and Kristein, M. D.: Changes in Blood Ketones During Fever, Proc. Soc. Exper. Biol. & Med. 45:640 (Nov.) 1940.
8. Paul, W. D., and Kemp, C. R.: Variations in the Concentration of Certain Electrolytes of the Blood Serum During Induced Hyperpyrexia, Proc. Soc. Exper. Biol. & Med. 45:427 (Oct.) 1940.
9. Osborne, S. L., and Farmer, C. J.: Influence of Hyperpyrexia on Ascorbic Acid Concentration in Blood, Proc. Soc. Exper. Biol. & Med. 49:575 (Apr.) 1942.

9. The pulse rate is increased. 10. Studies of the blood complement content, opsonins and phagocytic property of leukocytes reveal no changes; a slight increase in agglutinins may be noted with succeeding treatments.[10] 11. Capillary resistance is decreased,[11] with resultant capillary dilatation. 12. The basal metabolic rate is elevated, and there is a fall in oxygen tension.[12] 13. Excessive perspiration occurs, with loss of a great deal of chlorides from the body fluids. However, with adequate replacement of fluids and chlorides by mouth or vein, there is little change in blood volume. 14. The filling time of the ventricles is decreased with temperatures to 100.5 F. (38.1 C.).[13]

Methods

The patients in this study received fever therapy with general body temperatures elevated to 101 F. by means of a hypertherm cabinet. The sessions of fever were usually given on alternate days. After the patient's general body temperature had been elevated to the desired degree, he was removed from the cabinet and wrapped in a sheet and wool blanket and allowed to cool gradually until his body temperature had returned to normal.

In addition, all patients in this study were given other physical therapy procedures on the days they did not receive fever therapy; such procedures as massage, radiant heat, hydrotherapy, corrective exercise and occupational therapy were administered in a definitely coordinated manner. Each patient, after initial study, was fitted into this program in predefined outline of therapy, so adjusted as to benefit him to the greatest extent and to speed as rapidly as possible his recovery.

Most arthritic patients also show other clinical manifestations of their disease process; secondary anemia, fatigue, underweight, anorexia, increased irritability, insomnia and "the poor mental attitude of the chronically ill patient" were among those commonly encountered. Hence it was necessary first to instruct the patient so that he might better understand the share that he must play in his treatment. It was considered good psychology to place the new patient in a room with another patient who on admission had manifested the same "don't care" attitude and who was making excellent progress toward recovery. Talking over common problems of their disease and the phases of therapy were invaluable in the psychologic adjustment of the new patient. General procedures consisted in administration of ferrous sulfate for the secondary anemia or transfusions of whole blood if the anemia was severe; high caloric diets for the underweight; high vitamin therapy, especially factors of the vitamin B complex, for anorexia and sedatives for insomnia and to secure adequate rest; the salicylates combined with ascorbic acid were used to control muscle and joint pains. Calcium gluconate (10 cc. of 10 per cent solution) was given intravenously to control the root pains of the patients with spondylitis. Finally, as a necessary step, the entire staff maintained an attitude of optimism, which added to the reassurance of the patient group, who soon began to feel that their welfare and treatment were paramount in importance.

A group of selected patients with rheumatoid arthritis and rheumatoid arthritis and spondylitis in the series received gold in the form of gold sodium thiomalate. Gold was always administered intramuscularly and on the same day but before the patient received fever therapy. The dosage of the gold salt administered was the same as that recommended by Comroe[14] and Rawls[15]. 5 mg. twice weekly for two weeks, 10 mg. once a week for two weeks and thereafter 25 mg. weekly. Every patient receiving chrysotherapy was carefully watched for untoward reactions to the drug.

Results

This original group included 138 patients. For 103 of them it was felt that sufficient data had been accumulated from which the results could be adequately interpreted; the other 35 patients were therefore excluded from

10. Footnote deleted on proof.
11. Rossman, P. L.: Capillary Resistance in Artificially Induced Fever, Ann. Int. Med. 14:281 (Aug.) 1940.
12. Cullin, S. C.; Weir, E. F., and Cook, E.: The Rationale of Oxygen Therapy During Fever Therapy, Anesthesiology 3:120 (March) 1942.
13. Krusen, F. H.: Physical Medicine, Ed. 1, Philadelphia, W. B. Saunders Company, 1941, p. 59.
14. Comroe, B. I.: Arthritis, Ed. 1, Philadelphia, Lea & Febiger, 1944, p. 430.
15. Rawls, W. B.; Gruskin, B. J.; Ressa, A. A.; Dworzan, H. J., and Schruber, D.: Analysis of Results Obtained with Small Doses of Gold Salts in the Treatment of Rheumatoid Arthritis, Am. J. M. Sc. 207:528 (April) 1944.

the study. Of the group of 103 patients, 59 had rheumatoid arthritis, 29 had rheumatoid spondylitis, 7 had a combination of rheumatoid spondylitis and rheumatoid arthritis and 8 had gonorrheal arthritis.

The criteria for the evaluation of the severity of arthritis are inadequate and difficult to apply, since many arthritic patients not only have arthritis of one or multiple joints but manifest some other disease process concomitant with the arthritis, which of necessity modifies their treatment and retards their recovery. Again, a patient may have a severe deforming arthritis involving principally one joint, whereas another may have a relatively slight process involving multiple joints. Hence the patients were grouped according to pathologic classification: those with rheumatoid arthritis, rheumatoid arthritis combined with rheumatoid spondylitis, rheumatoid spondylitis and gonorrheal arthritis.

It is further realized that the criteria for the evalution of improvement of the arthritic patient are difficult to intepret. However, in this series the results were classified as (1) remission, (2) improved, and (3) unimproved.

A "remission" was recorded when the patient stated that there was relief of pain and stiffness, the degree of fatigue was less and it occurred less frequently. The objective evidence consisted of decreased swelling, increased motion of joints without pain, gain in weight, improvement of the secondary anemia, a better mental attitude, and usually a decrease in the blood sedimentation rate (Cutler method) to normal or close to normal. "Improved"

TABLE 1. — *Results of Treatment*

Diagnosis	Remission		Improved		Unimproved		Total	
	No.	%	No.	%	No.	%	No.	%
Rheumatoid arthritis	43	73	10	17	6	10	50	100
Rheumatoid spondylitis	18	62	6	20	5	18	29	100
Gonorrheal arthritis	8	100	....	....	--	....	8	100
Rheumatoid arthritis and rheumatoid spondylitis	2	28.5	3	43	2	28.5	7	100

was recorded when the patient stated that there was a decrease in pain and stiffness. The objective evidence consisted of some decrease in swelling, some increase in joint motion, an improvement in mental attitude and some gain in weight, with, as a rule, a tendency for the blood sedimentation rate to be lower. The rest of the patients were classified as "unimproved." The results are tabulated in table 1.

In the group 25 patients with rheumatoid arthritis and 4 with a combination of rheumatoid arthritis and rheumatoid spondylitis received chrysotherapy. The average dose of gold sodium thiomalate per patient was 240 mg. (120 mg. of gold). No reactions to the gold salts were observed in any patient of this group. Although the dose of gold sodium thiomalate was not large, it was felt that according to percentages expressed by other authorities[16] that reactions could be expected in from 7 to 15 per cent of patients receiving gold therapy. Hence, we should have expected from 1 to 3 patients to have some type of reaction to the gold; none occurred. Cohen, Goldman and Dubbs[17] reported untoward reactions in 10.3 per cent of 417 courses of

16. Comroe, B. I.: Arthritis, Ed. 1, Philadelphia, Lea & Febiger, 1944, pp. 440 and 444. Cecil, R. L.: The Problem of Dosage in the Administration of Gold Salts for Rheumatoid Arthritis, M. Clinic North America 30:545 (May) 1946. Freyberg, R. H.; Block, W. D., and Wells, G. S.: Gold Therapy for Rheumatoid Arthritis: Considerations Based Upon Studies of the Metabolism of Gold, Clinics 1:537, 1942.

17. Cohen, A.; Goldman, J., and Dubbs, A. W.: The Treatment of Rheumatoid Arthritis with 517 Courses of Gold, New England J. Med. 233:199 (Aug.) 2945.

gold treatment with Solangol-B Oleosum; in addition, they reported only 8.9 per cent untoward reactions in 259 patients receiving a single course of gold. However, in 96 patients receiving two courses of gold the percentage of untoward reactions rose to 15.6 per cent. There was one fatality in their series.

The probable explanation for the lack of untoward reactions to gold sodium thiomalate in this series is, first, the small dose of gold used and, second, to the use of fever therapy following the administration of the gold salts; and finally to the small number of patients in which gold therapy was used. It is felt that fever therapy, as used in this study, in combination with gold has definite merit in that the marked increase in blood flow and capillary dilatation afford better distribution of the injected gold salts[18] and aid in the prevention of untoward reactions. Table 2 tabulates the results in patients receiving gold in this stundy.

There was a slight decrease in percentage of remissions and improvements in the group receiving gold therapy. However, this is explained on the basis that the more severely involved patients were selected to receive chrysotherapy.

A few general statistics of this study reveal that there were 1,936 low grade fever therapy sessions administered, an average of 19.7 sessions per patient. If this figure is further broken down, it is found that the average number of low grade fever therapy sessions for patients with rheumatoid

TABLE 2. — *Results for Patients Receiving Gold Sodium Thiomalate*

Diagnosis	Remission		Improved		Unimproved		Total	
	No.	%	No.	%	No.	%	No.	%
Rheumatoid arthritis	17	68	5	20	3	12	25	100
Rheumatoid arthritis and rheumatoid spondylitis	2	50	0	0	2	50	4	100

arthritis was 17.4; for the patients combined rheumatoid arthritis and rheumatoid spondylitis, 21.9; for patients rheumatoid spondylitis, 15.4 and for patients with gonorrheal arthritis, 20.7. The average number of days of treatment per patient was 69.8 for those with rheumatoid arthritis, 56.4 for those with combined rheumatoid arthritis and spondylitis; 86.7 for those with spondylitis, and 79 for those with gonorrheal arthritis.

Comment

In this study 90 per cent of the patients with rheumatoid arthritis showed a remission or improvement; this is roughly in accord with the findings of others.[17] In the group with rheumatoid spondylitis there were 82 per cent who manifested a remission or improvement, and in the group with combined rheumatoid arthritis and rheumatoid spondylitis 71.5 per cent showed a remission or improvement. The remissions were by far smaller in this group. In the gonorrheal arthritis group 100 per cent had remissions; all but one of these patients were in the chronic phase of the disease. The one in the acute phase received a combination of penicillin and low grade fever therapy.

Kendell[19] has stated that the concurrent use of chemotherapy with artificial fever therapy increases the therapeutic efficiency of both agencies.

18. Drewyer, G. E., and Hughes, J .E.: A New Approach to the Problem of Incipient and Recurrent Malaria, Arch. Phys. Therapy 25:273 (May) 1944.

19. Kendell, H. W.; Simpson, W. M., and Rose, D. L.: Combined Artificial Fever-Chemotherapy in the Treatment of Neurosyphilis, Ohio State Med. J. 38:909 (Oct.) 1942.

Hench[20] concluded that fever therapy may benefit arthritis by (1) direct bacteriolytic or bacteriostatic effects due to an influence of heat on the bacteria; (2) a local effect from vasodilatation, providing an augmented blood supply to inflamed tissues; (3) an indirect increase in the formation or mobilization of immune bodies, and (4) a general effect from the heightened metabolism incident to fever. However, recently Hench[21] has concluded that the use of hyperpyrexia with high temperatures over prolonged periods is of no value in the treatment of arthritis and, in fact, is contraindicated.

From my observations, low grade fever therapy as here defined merits further study. The rapid absorption of joint exudates, the decrease in synovial thickening, the diminution of pain, the gain in weight, the improved mental attitude, and the early ambulation of bedridden patients are some of the improvements noted early with the use of low grade fever therapy. It is my impression that arthritic patients are aided by low grade fever therapy because of the better circulation of blood, greater capillary dilatation and improved metabolism. Such physiologic changes aid in the better distribution of the drugs used, plus a better absorption of food from the intestinal tract, and an increase in blood flow through inflamed tissues. In addition, it is my opinion that low grade fever therapy apparently decreases the number of untoward reactions when gold therapy is used.

In organizing the treatment of a patient with arthritis the first essential is a complete history and physical examination, including such laboratory studies as blood count, determination of blood sedimentation rate and Kahn reaction, urinalysis and other laboratory procedures indicated. All of these are essential for proper evaluation of the type of arthritis. After completion of these studies a broad plan of therapy for the individual patient can be outlined. Such a plan should include the necessary medications for the comfort of the patient, adequate diet and vitamin intake, transfusions of whole blood if indicated, and physical therapy as indicated in order to facilitate his recovery and secure proper joint function or to maintain as much joint function as is possible. If gold therapy is indicated, as it is for certain patients with rheumatoid arthritis, its place in the broad plan of treatment should be provided for. Such a comprehensive survey of the individual patient and planning of his therapy before starting will often preclude poor results.

Early ambulation of the bedfast patient is essential. Joint motions and function must be maintained, and, after observation, this can best be accomplished by active motion on the part of the patient, rather than passive motion of the joint by the physical therapist. Active motion in gradually increasing amounts can be accomplished in spite of a slight increase in pain. It is one of the best mental tonics that can be given the bedridden arthritic patient. Prolonged sessions of hyperpyrexia with general body temperature elevated above 101 F. are too debilitating to the patient and are definitely contraindicated, in my opinion.

When gold therapy is contemplated, small doses of gold salts should be used. It is seldom necessary to exceed 50 mg. as a weekly dose, and in my experience equally good results can be obtained when the maximal weekly dose does not exceed 25 mg. The administration of gold salts should be combined with low grade fever therapy, as this combination seems to give better distribution of the gold and aids in the elimination of untoward re-

20. Hench, P. S.: The Present Status of Fever Therapy in the Treatment of Gonorrheal Arthritis, Chronic Infection (Atrophic) Arthritis and Other Forms of Rheumatism, J. Lab. & Clin. Med. 21:524, 1936.
21. Hench, P. S.: Personal communication to the author, 1947.

TABLE 3. — *Summary*

Case No.	Age	Fever Therapy No. of Sessions	Gold Sodium Thiomalate, Mg.	Duration of Treatment, Days	Sedimentation Rate Before	Sedimentation Rate After	Result
Gonorrheal Arthritis							
1	21	30	0	91	19	3	Remission
2	21	21	0	55	16	6	Remission
3	22	9	0	98	16	2	Remission
4	21	9	0	91	16	4	Remission
5	27	21	0	86	26	4	Remission
6	23	22	0	57	23	2	Remission
7	36	36	0*	56	29	3	Remission
8	21	18	0	99	22	4	Remission
Rheumatoid Arthritis							
9	32	10	0	137	20	21	Unimproved
10	41	9	0	51	7	3	Remission
11	29	12	0	31	18	12	Improved
12	23	5	0	17	15	2	Remission
13	21	8	0	24	16	3	Remission
14	38	13	90	44	8	3	Remission
15	32	9	0	23	15	10	Remission
16	17	8	0	35	20	8	Remission
17	30	17	215	94	24	19	Unimproved
18	24	21	0	62	18	12	Improved
19	39	12	0	109	20	21	Unimproved
20	31	25	215	81	17	9	Remission
21	37	16	125	62	20	13	Remission
22	18	28	0	70	20	7	Remission
23	37	10	0	44	17	6	Remission
24	36	14	0	55	19	11	Remission
25	32	13	155	107	16	11	Unimproved
26	34	15	75	110	26	7	Remission
27	33	6	0	31	17	7	Remission
28	24	49	340	186	27	14	Remission
29	26	10	0	43	18	16	Improved
30	32	8	0	54	8	2	Remission
31	22	9	0	29	12	8	Remission
32	19	10	0	36	14	2	Remission
33	21	7	0	31	21	6	Remission
34	43	13	0	36	27	9	Remission
35	31	19	0	49	28	21	Improved
36	25	10	0	45	22	13	Remission
37	40	24	0	73	18	15	Improved
38	38	37	165	128	18	13	Remission
39	35	10	90	95	22	9	Remission
40	38	22	195	67	25	8	Remission
41	45	21	0	61	21	3	Remission
42	40	24	140	77	27	23	Remission
43	26	27	225	92	22	12	Improved
44	22	6	0	54	12	5	Remission
45	38	14	0	45	15	10	Remission
46	25	15	115	47	23	10	Remission
47	21	11	0	91	14	8	Remission
48	21	12	0	35	18	7	Remission
49	34	7	0	23	12	5	Remission
50	19	23	290	100	25	14	Remission
51	20	14	0	40	20	7	Remission
52	23	18	290	131	25	19	Remission
53	26	24	165	63	23	14	Remission
54	27	15	105	48	23	9	Improved
55	23	12	90	43	18	3	Remission
56	39	20	140	62	23	18	Remission
57	19	38	340	128	28	15	Unimproved
58	27	28	250	84	31	17	Remission
59	29	19	0	60	14	6	Remission
60	26	46	490	149	31	21	Improved
61	20	26	215	76	26	19	Improved
62	34	15	0	64	22	10	Unimproved
63	39	14	0	44	18	7	Remission
64	33	13	0	36	21	5	Remission
65	42	27	322.5	180	26	12	Remission
66	59	10	90	45	24	16	Improved
67	24	49	0	186	26	14	Remission

* This patient received 3,500,000 units of penicillin (20,000 units intra-articularly).

Case No.	Age	Fever Therapy No. of Sessions	Gold Sodium Thiomalate, Mg.	Duration of Treatment, Days	Sedimentation Rate Before	Sedimentation Rate After	Result
Spondylitis							
68	33	9	0	40	14	3	Remission
69	20	14	0	42	22	14	Remission
70	38	18	0	58	23	12	Remission
71	18	17	0	61	24	20	Unimproved
72	28	30	0	94	20	9	Improved
73	30	18	0	69	15	3	Improved
74	36	38	0	139	24	11	Remission
75	26	10	0	61	22	10	Remission
76	33	16	0	61	20	19	Unimproved
77	30	11	0	40	10	6	Remission
78	23	20	0	71	22	9	Improved
79	34	21	0	75	25	19	Unimproved
80	24	26	0	122	22	17	Remission
81	27	18	0	66	17	6	Remission
82	32	34	0	125	23	13	Remission
83	24	20	0	85	23	3	Remission
84	35	26	0	102	22	9	Remission
85	32	14	0	66	22	13	Remission
86	24	49	0	169	24	6	Improved
87	29	23	0	123	25	12	Remission
88	22	7	0	32	15	7	Remission
89	18	17	0	97	12	5	Remission
90	35	42	0	183	23	21	Unimproved
91	33	21	0	68	17	3	Remission
92	30	52	0	177	22	11	Unimproved
93	26	8	0	38	20	15	Improved
94	40	15	0	86	21	14	Improved
95	33	17	0	68	17	13	Remission
96	26	24	0	87	19	8	Remission
Spondylitis and Rheumatoid Arthritis							
97	23	11	55	49	16	11	Unimproved
98	26	18	140	59	29	18	Unimproved
99	29	26	0	81	23	15	Improved
100	31	14	0	58	21	13	Improved
101	30	21	0	68	23	12	Improved
102	34	9	65	19	30	17	Remission
103	42	19	140	61	30	19	Remission

actions. The amount of gold to be given in an individual case is best judged by the response of the patient. To illustrate, if a remission occurs with 165 mg., small doses of gold should be given at bimonthly or monthly intervals for several years in order to keep the patient in remission with the hope of a permanent cure. In my opinion, this is the method of choice rather than the arbitrary method of administering gold in courses.

Finally, it should be pointed out that there are several reasons for the failure of many arthritic patients to manifest satisfactory improvement; first, the presence of some other disease in addition to the arthritis, such as chronic pulmonary infections, chronic renal disease, rheumatic heart disease, obesity, chronic colitis, and a host of others; second, failure on the part of the attending physician to plan an adequate course of therapy to meet the needs of the individual arthritic patient in facilitating his recovery, and third, failure on the part of the patient to cooperate to the fullest extent in the program outlined for his recovery. Finally, there are certain arthritic patients whose disease process seems to progress steadily in spite of what seems adequate therapy, but this is equally true in other disease processes to which man is subjected.

Summary

1. A brief enumeration of the physiologic principles of fever therapy has been presented.

2. A study of 103 cases of various types of arthritis treated low grade fever therapy, plus a broad medical program, individualized for each patient

to facilitate his recovery, has been presented. The results obtained have been reviewed.

3. A few general principles in the treatment of arthritis have been stated.

Discussion

Dr. William Bierman (New York): The results of the treatment of rheumatoid arthritis with or without spondylitis as reported by Dr. Drewyer are extraordinarily good. He employed a combination of treatments, much as chemotherapy (particularly gold), psychotherapy and various forms of physical therapy, in addition to elevation of systemic temperature. It therefore becomes difficult to evaluate the importance of any one of these procedures. The idea of combining gold and fever therapy is an important contribution to the care of a condition so widespread and so difficult to treat as rheumatoid arthritis. Our own results and those reported by others employing fever therapy alone are not at all as good.

Dr. Drewyer has referred briefly to the literature describing the physiologic changes produced by the elevation of body temperature through physical means. A more comprehensive reference to the literature on this phase of the subject would have required a greatly extended presentation, if not, indeed, a large paper on this subject alone. The question arises also as to how much this consideration of altered physiology is pertinent to the main subject of the paper, inasmuch as most of these physiologic alterations have been reported as occurring as a result of temperature elevations considerably beyond 101 F.

The reference to cases of chronic gonorrheal arthritis raises the question as to whether higher temperatures, with or without the use of penicillin, would have brought about cures in a much shorter time.

SOME ENZYME SYSTEMS OF DENERVATED MUSCLE *

ERNST FISCHER, M.D.

RICHMOND, VA.

In previous papers[1] a colleague and I reported that after denervation of rabbit gastrocnemius muscles, myosin — the main protein of the muscle and the probable substrate of the contractile mechanism — decreases considerably with progressing atrophy, and that, like the weight loss, this deterioration of the muscle protein could be retarded distinctly by appropriate electrical stimulation. Engelhardt and Ljubinova's discovery[2] in 1939 that myosin has strong adenosine triphosphatase activity and that the muscle adenosine triphosphatase is either myosin or a protein closely associated with myosin has been more or less confirmed in the meantime by various independent investigators.[3] Kalckar[4] and Szent-Györgyi[5] developed theoretical conceptions of the fundamental mechanism of muscular contraction based upon this close association between myosin and adenosine triphosphatase. It was, therefore, of interest to investigate whether the myosin, which diminishes quantitatively during atrophy, loses adenosine triphosphatase activity also. It was especially important to find out whether experimental evi-

* From the Basic Research Division, Baruch Center of Physical Medicine, Medical College of Virginia. This work was done under contract between the Office of Naval Research and the Medical College of Virginia. The various phases of these investigations were performed in collaboration with Dr. Ernst Huf or Mr. Russell V. Bowers and with the technical assistance of Virginia W. Ramsey, Kathleen W. Ryland, Gerda L. Nothmann and Paul E. Totten.

* Read at the Twenty-Fifth Annual Session of the American Congress of Physical Medicine, Minneapolis, Sept. 6, 1947.

1. Fischer, E., and Ramsey, V. W.: (*a*) Am. J. Physiol. **145**:571, 1946; (*b*) Am. J. Physiol. **145**: 583, 1946.
2. (*a*) Engelhardt, W. A., and M. N. Ljubimova: Nature **144**:668, 1939; (*b*) Engelhardt, W. A.: Yale J. Biol. & Med. **15**:21, 1942.
3. Szent-Györgyi, A., and Banga, I.: Science **93**:158, 1941. Needham, D. M.: Biochem. J. **36**: 113, 1942. Bailey, K.: ibid. **36**:121, 1942. Ziff, M., and Moore, D. H.: J. Biol. Chem. **153**:653, 1944. Singher, H. O., and Meister, A.: ibid. **159**:491, 1945.
4. Kalckar, H. M.: J. Biol. Chem. **148**:127, 1943.
5. Szent-Györgyi, A.: Bull. Soc. chim. Biol. **25**:242, 1943.

dence could be found to support the following working hypothesis. In denervation atrophy, the adenosine triphosphatase activity diminishes because of disuse of this enzymatic system finally leading to deterioration of the myosin. If the adenosine triphosphatase activity is kept up by enforced use of the enzyme system by electrically evoked contractions, the myosin deterioration is prevented.

Wartime research by Szent-Györgyi and co-workers,[6] which became known in this country only in 1946, made it obvious that the protein which we described in our previous publication[1] as "myosin" was in reality actomyosin, a combination of myosin with actin, another specific muscle protein, described first by Straub.[7] Actin has no adenosine triphosphatase activity, and the activity of actomyosin depends on the varying amount of myosin.

Therefore, we repeated our previous experiments, isolating now a more or less pure myosin according to the method of Szent-Györgyi[5] by extraction with 0.6 M potassium chloride at 0° for only fifteen minutes, while the actomyosin in the earlier experiments was extracted with 0.6 M lithium chloride for one hour at room temperature. By precipitating and twice redissolving and reprecipitating with twenty times the volume of distilled water at a

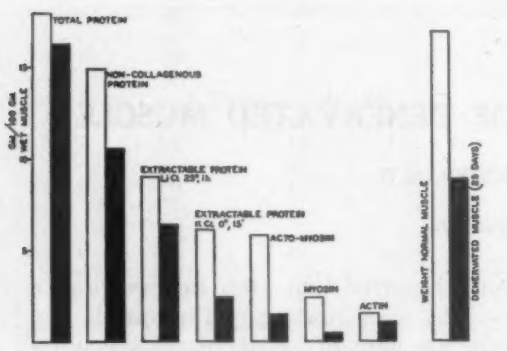

Chart 1. — Comparison of protein data for normal rabbit gastrocnemius muscles and gastrocnemius muscles denervated twenty-five days previously. All data are averages for at least seven independent determinations and are measured as grams of protein per 100 gm. of wet muscle.

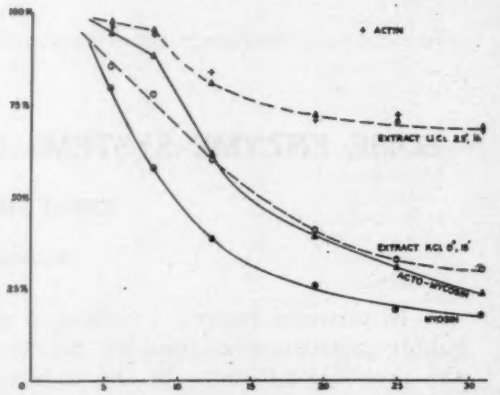

Chart 2. — Changes in denervated muscles expressed in per cent of corresponding values for normal muscles. Each point represents the average of at least five independent determinations. Time since denervation in days.

pH of 6.9, pure myosin and actomyosin were obtained from the two crude extracts, respectively. In the myosin series, the actin content also was determined by Straub's method.[7] That the results of these two independent series were comparable is attested by the fact that the average values for total protein, noncollageneous protein, nonprotein nitrogen and weight are identical for normal muscles and for muscles denervated for the same length of time.

Chart 1 shows a comparison between protein data for normal muscle and for muscles denervated for twenty-five days. The data of this figure are the averages for groups of at least seven experiments. All data are calculated as grams per hundred grams of wet muscle weight. For normal muscle, the difference between the crude lithium chloride extract and the crude potassium chloride extract is larger than corresponds to the amount of actin determined according to Straub's method. This is probably due to the fact that during the short extraction in the cold not all the myosin

6. Szent-Györgyi, A.: Studies on Muscle, Acta physiol. Skandinav. 9: sup. 25, 1945; Chemistry of Muscular Contraction, New York Academic Press, 1947.
7. Straub, F. B.: Stud. Inst. Med. Chem. Univ. Szeged 2:3, 1942.

can be dissolved. It is further obvious from those data that a relatively larger fraction of purified actomyosin can be gained from the lithium chloride extract than myosin from the potassium chloride extract. Denervation of twenty-five days decreases the actin content and the lithium chloride protein extract in an amount corresponding only to the small decline in non collagenous protein, whereas the potassium chloride extract is much more diminished. The fractions obtainable as actomyosin and myosin, respectively, are decreased much more than their respective crude extracts. This means that in the extracts of denervated muscles, a smaller part of the proteins present is precipitated by the conditions typical for actomyosin and myosin.

The time course of the protein changes during denervation is presented in chart 2. The loss in actin corresponds to the loss in lithium chloride — extractable protein. However, the amount of actomyosin obtainable from

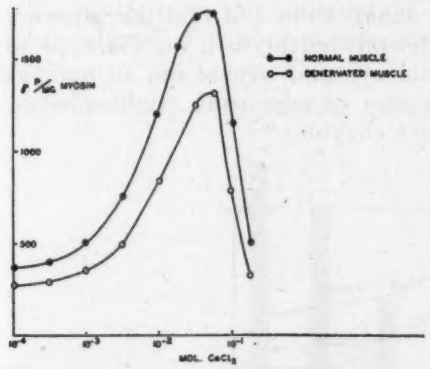

Chart 3. — Influence of $CaCl_2$ concentration upon the adenosine triphosphatase activity of myosin from normal and from denervated muscle (fourteen days). Activity measured as micrograms of phosphorus liberated per milligram of protein at 38 C. in six minutes.

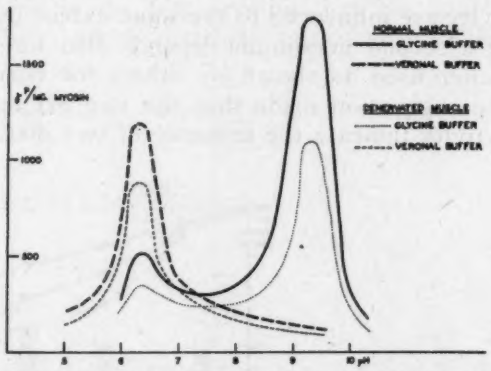

Chart 4. — Influence of pH and type of buffer used upon the adenosine triphosphatase activity of myosin from normal and from denervated muscle (twenty-four days). Activity measured as micrograms of phosphorus liberated per milligram of protein at 38 C. in six minutes.

these extracts decreases much more rapidly, since with progressive atrophy the ratio between precipitable and extractable protein decreases. The loss in potassium chloride — extractable protein is quicker than that in lithium chloride — extractable protein, and again the precipitated myosin declines more than the crude extract. That actin declines only little and myosin much more than actomyosin indicates that with progressing atrophy the ratio between the actin and the myosin forming the actomyosin compound increases. The increase in this ratio is probably the explanation for the changes in some physicochemical properties of the actomyosin with progressing atrophy which we reported a few years ago.[8]

Preliminary experiments have shown that the lithium chloride extracts tend to have about the same adenosine triphosphatase activity calculated per original gram of muscle as the lithium chloride extracts, which contain less protein. Therefore, we restricted our enzymatic studies to the potassium chloride extract and myosin. We soon observed that in contrast to normal myosin, which gives maximal adenosine triphosphatase activity after three or — at the most — four precipitations, the activity of myosin from denervated muscle increases with further precipitation. However, it is impossible to repeat the purification procedures too often, since (1) the amount of myosin obtainable from one denervated gastrocnemius muscle soon be-

8. Fischer, E., and Ramsey, V. W.: Arch. Phys. Therapy 25:709, 1944.

comes so small that quantitative studies become very difficult and (2) the time needed to repeat these procedures so often is so long that normal myosin submitted to them finally shows a decrease in activity. Although we did not succeed in obtaining myosin from denervated muscles with the same activity as that from the control muscle, we feel that if purification could be hastened, even a muscle denervated for five weeks would yield a very small fraction of myosin of about normal adenosine triphosphatase activity.

In order to be able to compare quantitatively the adenosine triphosphatase activity of normal and denervated muscle, we worked finally — for practical reasons only — with the crude extract and thrice precipitated myosin. The influence of electrolytes upon the adenosine triphosphatase activity of pure myosine from denervated muscles corresponds exactly to that described for normal muscle.[2b] In both chart 3 and chart 4, the activity is calculated per milligram of protein present, and it is obvious from these curves that myosin from denervated muscle has a lower adenosine triphosphatase activity. The fact that in *p*H experiments, the maxima of the activity are influenced to the same extent by denervation and that the presence of a second maximum depends also for denervated myosin on the type of buffer used as shown by others for normal myosin,[9] seems not to support the suggestion made that the two *p*H maxima of adenosine triphosphatase activity indicate the presence of two distinct enzymes.[10]

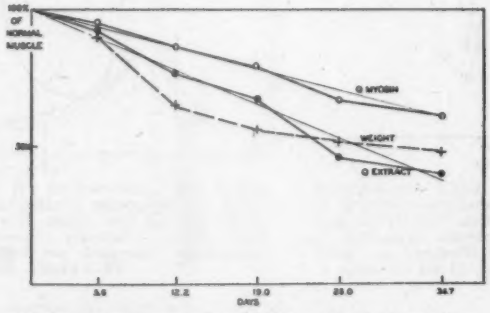

Chart 5. — Changes in muscle weight and in adenosine triphosphatase activity per milligram of protein of KCl extract and myosin from denervated muscles expressed in per cent of the values for the control muscles. Each point represents the average of at least five independent determinations. Time since denervation in days.

The time course of the diminution of the adenosine triphosphatase activity of the crude extract and the myosin is shown in chart 5. All enzymatic assays were done with two different enzyme concentrations inside the linear range of enzymatic rate. For comparison, the enzymatic rate Q was calculated as micrograms of phosphorus liberated per milligrams of protein at 38 C. in six minutes and then the Q of the denervated material eypressed in per cent of the activity of the normal one. Both Q's diminish with progressing atrophy at a roughly constant rate but Q myosin at a distinctly lower rate than Q extract. The larger decline of the latter is an expression of the progressing decrease of the ratio between precipitable myosin and total protein content of the extract. Chart 5 contains the weight curve also, and it is evident that the decrease in adenosine triphosphatase activity does not precede the weight loss. However, this result does not invalidate our working hypothesis yet, because this curve of loss in adenosine triphosphatase

9. Mommaerts, W. F. H. M., and Scraidarin, K.: J. Gen. Physiol. 30:401, 2947.
10. Meister, A.: Science 106:167, 1947.

activity does not take into consideration the progressive decrease of the amount of myosin present.

If one assumes that in the extracted muscle mash no adenosine triphosphatase activity is retained or that at least the ratio between retained and extracted activity is not changed by atrophy, one can calculate from our data the relative values of the total activity per gram of muscle. This can be done by using either the Q and protein concentration values for the extracts or the corresponding values for the myosins. Chart 6 represents the results of such calculations and shows that for both normal and atrophied muscles the adenosine triphosphatase activity per gram of muscle is higher when calculated from the extract data. The ratio between Q muscle from myosin data and Q from extract data declines at a constant rate with atrophy. This indicates probably that with progressing atrophy a larger fraction of the adenosine triphosphatase is no longer absorbed on the myosin. We determined in a few experiments the activity of the supernatant fluid and of the precipitated myosin after each precipitation. With myosin from normal muscle, only the supernatant fluid of the first precipitation has appreciable adenosine triphosphatase activity, which corresponds probably to the

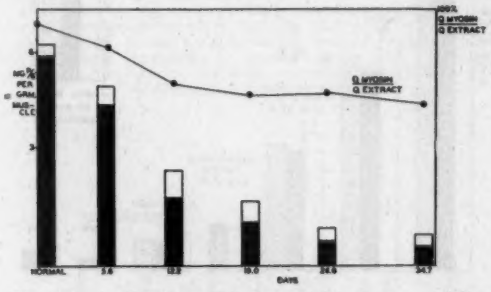

Chart 6. — Relative adenosine triphosphatase activity per gram of muscle calculated from the extract data (white columns) and from the myosin data (black columns) in milligrams of phosphorus liberated at least five independent determinations. Time since 38 C. in six minutes. All data are averages for at denervation in days.

water-soluble adenosine triphosphatase of muscle described first by Lohmann.[11] With myosin from denervated muscle, however, with each further precipitation considerable adenosine triphosphatase activity is still retained in the supernatant fluid. This myocin behaves, therefore, rather similarly to normal myosin after fractionation and probably partial denaturation by lanthanium salt.[12]

We interpret our results that with progressing atrophy some of the myosin changes and forms a much more stable and inactive combination with actin, so that only a small fraction can be extracted and precipitated as myosin, but even this myosin is somewhat deteriorated, as demonstrated by the diminished adenosine triphosphatase activity. If our assumption is correct that the activity retained in the muscle mash is proportional to the extracted enzymatic activity, the loss of total adenosine triphosphatase activity precedes and is quicker than the weight loss, which would support our working hypothesis. The fact that denervated myosin loses adenosine triphosphatase activity with purification suggests that the procedure used, which do not influence normal myosin, in deteriorated myosin weaken the

11. Lohmann, K.: Biochem. Ztschr. 271:264, 1934.
12. Polis, B. D., and Meyerhof, O.: J. Biol. Chem. 169:389, 1947.

links between the protein skeleton of the myosin and the active adenosine triphosphatase molecules; the latter, one may classify as "protein" [13] as does Szent-Györgyi.

According to our hypothesis, appropriate electrical treatment should retard these myosin changes. Chart 7 demonstrates that such treatment has considerable effect, as shown by various authors,[14] on the weight of the denervated muscles, especially if the muscles contract during stimulation, not freely but against heavy resistance.[15] However, the effect upon the protein changes is much less distinct, although, per gm. muscle, the increases in total protein, noncollagenous protein, lithium chloride — extractable protein, actomyosin and myosin are statistically significant, at least for the so-called isometric treatment. Although the effect of this treatment on the protein concentration is small, it nevertheless, as shown in chart 8, improves considerably the adenosine triphosphatase activity of the purified myosin and to a lesser extent that of the extract. If the relative adenosine triphos-

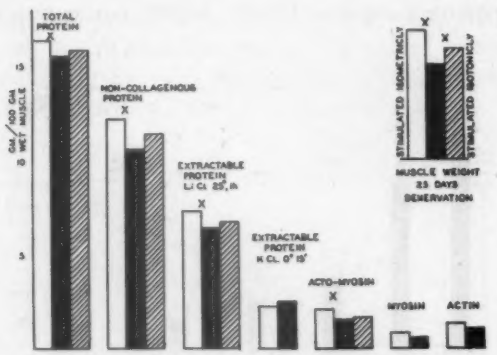

Chart 7. — Comparison of protein data for denervated control muscles and denervated muscles treated with daily electrical stimulation, either the muscles contracting against heavy resistance (isometrically) or shortening freely (isotonically). All muscles denervated for twenty-five days. All data are averages for six independent determinations. Crosses between columns designate the differences as statistically significant.

phatase activity per gram of muscle is calculated for the treated muscle, the ratio between activity calculated from myosin data and that from extract data approaches the ratio found for normal muscles.

But the total activity per gram of treated muscle is still only a fraction of that of normal muscle. This series of experiments apparently offers no support to our hypothesis. However, one factor interferes in these experiments and makes the effect of stimulation upon the adenosine triphosphatase activity look less pronounced than it might be in reality. Since Deuticke's first report, it is well known that electrical stimulation diminishes the solubility of the muscle proteins.[16] To minimize this effect, we killed our animals only twenty-four to thirty-six hours after the last treatment. As seen from chart 7, the fact that the potassium chloride-extractable protein is not

13. Banga, I.; Guba, F., and Szent-Györgyi, A.: Nature 149:1947.
14. Fischer, E.: Am. J. Physiol. 127:605, 1939. Gutmann, E., and Guttmann, L.: J. Neurol., Neurosurg. & Psychiat. 7:7, 1944. Jackson, S.: Brain 68:300, 1945. Hines, H. M.; Melville, E., and Wehrmacher, W. H.: Am. J. Physiol. 144:278, 1945. Kosman, A. J.; Osborne, S. L., and Ivy, A. C.: Arch. Phys. Med. 28:7 and 12, 1947.
15. Hines, H. M., and Wehrmacher, W. H.: J. Iowa M. Soc. 34:142, 1944. Fischer. 1b.
16. Deuticke, H. J.: Arch. f. d. ges. Physiol. 224:1, 1930; Ztschr. f. Physiol. Chem. 210:97, 1932. Kamp. E.: Biochem. Ztschr. 307:226, 1941; Dubuisson, M., and Jacob, J.: Bull. Soc. roy. sc. Liége 3:146, 1945; Mrazek, R. G., Jr., and Reid, C. I.: Am. J. Physiol. 149:177, 1947.

increased, but even somewhat decreased, by the treatment, induced us to investigate this problem by subjecting normal muscles to exactly the same treatment.

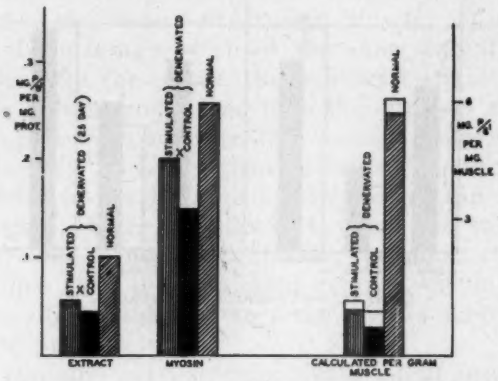

Chart 8. — Comparison of adenosine triphosphatase activity of KCl extract and myosin measured as micrograms of phosphorus per milligram of protein at 38° C. in six minutes and comparison of relative adenosine triphosphatase activity per gram of muscle for normal muscles, denervated muscles (twenty-five days) and isometrically treated denervated muscles (twenty-five days). All data represent averages of six independent determinations.

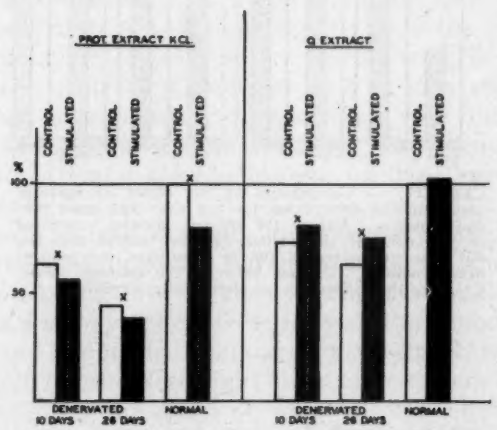

Chart 9. — Comparison of the effect of electrical treatment upon muscles denervated for ten and twenty-five days and upon normal muscles. Values for treated muscles expressed in per cent of the values for the untreated control muscles. All data are averages for at least five independent determinations. Crosses between columns designate the differences as statistically significant.

Chart 9 demonstrates that our treatment decreases potassium chloride-extractable protein considerably in normal muscle without increasing Q extract. Since the extract is reduced in normal muscle, it is not surprising that myosin is decreased to about the same extent in normal muscle (chart 10). But myosin in the treated denervated muscle is significantly increased. Q myosin in the normal muscle is insignificantly decreased, while the treated denervated muscle shows the typical increase in Q myosin.

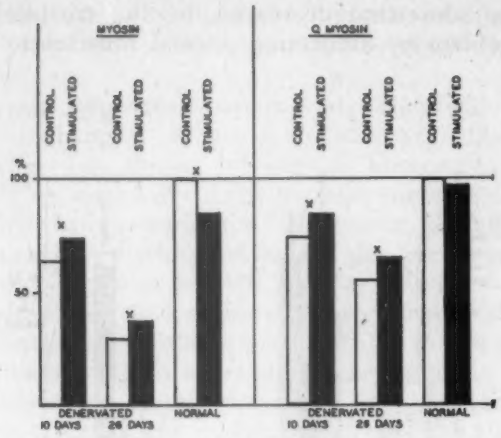

Chart 10. — Continuation of chart 9.

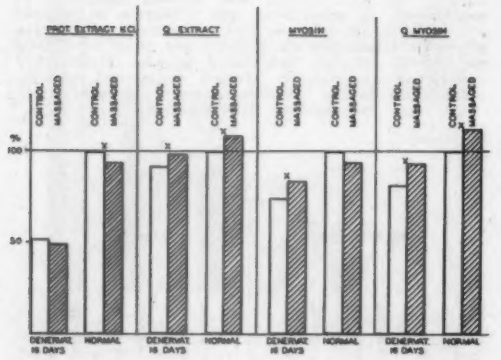

Chart 11. — Comparison of the effect of massage upon muscles denervated for ten days and upon normal muscles. Values for treated muscles expressed in per cent of the values for the control muscles. All data are averages for at least five independent determinations. Crosses between the columns designate the differences as statistically significant.

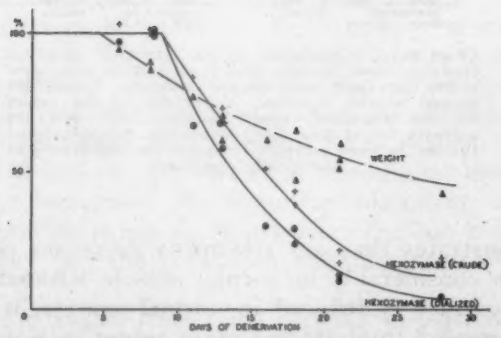

Chart 12. — Loss in muscle weight and changes in zymohexase activity of extracts from denervated muscles. All data expressed in per cent of the values for the normal control muscles. Each point represents the average of double determinations for the same muscle. Time since denervation in days.

Hines and co-workers[17] have demonstrated that massage increases the strength of denervated muscles without significantly affecting their weight. Therefore, we included massage in the investigation (chart 11) and found that massage also decreases potassium chloride-extractable protein somewhat in normal and in denervated muscles and that myosin from normal muscle is accordingly diminished, whereas more myosin can be obtained from the diminished extract of the treated denervated muscle. Q extract and Q myosin are increased by massage to about the same extent in normal and in denervated muscle. On the whole, the beneficial effect of massage on the denervated muscle is less pronounced than the effect of electrical treatment.

These last experiments indicate that in our main series the improvement in Q muscle must be larger than shown by our data, since, owing to the secondary effects of the treatment, less protein can be extracted. The fact that, nevertheless, larger amounts of pure myosin can be obtained from the extracts of treated denervated muscles and that these myosins have hiyher Q's, indicates that the treatment retards the myosin deterioration and the weakening of the links between the myosin skeleton and the adenosine triphosphatase proteins.

The literature contains only scattered data about other enzyme activity in denervated muscle. These data are not indicative that any relation exists between the change in one specific enzymatic activity and the course of atrophy. Reoxidation capacity is reduced early after denervation but after the second or third week decreases only slowly or even increases again.[18] Glutation is reported as increasing steadily with denervation,[19] whereas in succino-oridase and hydroginases only rather small changes were observed.[20]

Much need exists to investigate during denervation atrophy the changes in concentration of those enzymes involved in the main chemical reactions coupled with muscle activity. We started recently the investigation of the zymohexase, the emzyme responsible for the splitting of hexosediphosphate into two molecules of triosephosphates and in the resynthesis of hexosediphosphate from triosephosphates, processes very important in supplying the ultimate energy for muscle activity. The identity of hexozymase with the well known water-soluble muscle protein "myogen A" has been demonstrated recently.[21]

The preparation of the enzyme and the enzymatic assays were performed with methods modified for our purposes from the methods used by Herbert and co-workers.[22] As in the experiments with adenosine triphosphates we compared the zymohexase activity of the denervated muscles with activity of the normal controls. As shown in chart 12, zymohexase activity is not altered at all for the first eight days after denervation and is, therefore, clearly preceded by the onset of weight loss. There is apparently no relation between the course of weight loss and the diminution in zymohexase activity. How far electrical treatment influences the diminished zymohexase activity we have not yet investigated.

Although up to now we have been able to demonstrate only that adenosine triphosphatase activity diminishes at least as early as the weight loss in denervation atrophy, the fact that the decrease in this enzyme activity is distinctly faster than the weight loss makes it at least possible that the level of the adenosine triphosphatase activity is a determining factor in denervation atrophy. That the retardation of the weight loss by electrical treatment is accompanied with a distinct retardation of the loss in adenosine

17. Suskind, M. I.; Hajek, N. M., and Hines, H. M.: Arch. Phys. Med. 27:133, 1946.
18. Date, S.: Nagasaki Igakkai Zassi 9:959, 1931. Maschesi, F.: Riv. di pat. sper. 9:374, 1932. Knowlton, G. C., and Hines, H. M.: Am. J. Physiol. 111:253, 1934.
19. Kamiya, T.: Nagoya J. M. Sc. 5:18, 1930. Okuda, M. J.: J. Biochem. 11:183, 1930. Schéda, A.: Magyar orvosi, Arch. 36:327, 1935.
20. Levine, R.; Hechter, O., and Soskin, S.: Am. J. Physiol. 132:336, 1941.
21. Taylor, J. F.; Green, A. A., and Cori, G. T.: Federation Proc. 5:158, 1946.
22. Herbert, D.; Gordon, H.; Subrahmanyan, and Green, D. E.: Biochem. J. 34:1108, 1940.

triphosphatase activity, although this retardation could not be measured to its full extent, gives further support to our working hypothesis mentioned in the beginning. However, much further work is needed before the exact role of the various enzymatic reactions for denervation atrophy and its retardation will be known.

The discussion of this article will be published in a later issue of the ARCHIVES.

PHYSICAL MEDICINE AT A. M. A.

Special Exhibit on Physical Medicine

The Special Exhibit on Physical Medicine includes Physical Therapy, Occupational Therapy and Rehabilitation. It is presented for the third time under the auspices of the following committee:

Frank H. Krusen, Rochester, Minn., chairman.
Winfred Overholser, Washington, D. C.
Howard A. Rusk, New York.

The subjects covered in the exhibit, with the demonstrators in charge, are as follows:

J. F. HERRICK and E. J. BALDES, Mayo Foundation, Institute of Experimental Medicine, Rochester, Minn.:

Ultrasonics in Medicine: Devices for producing high frequency sound waves will be shown and the possibilities for use of these waves in medicine will be indicated.

K. G. WAKIM and D. J. ERICKSON, Sections on Experimental Physiology and Physical Medicine, Mayo Clinic, Rochester, Minn.:

Electrical Stimulation of Muscles: The results of experimental studies on electrical stimulation of muscles will be demonstrated and devices for this purpose will be shown.

O. LEONARD HUDDLESTON and JAMES G. GOLSETH Department of Physical Medicine, University of Southern California School of Medicine, and Los Angeles County General Hospital, Los Angeles:

Clinical Electromyography: The exhibit will consist of electromyograms, charts, drawings and photographs dealing with various clinical findings in neuromuscular disorders using electromyography. There will also be demonstrations of the procedure on various types of neurologic cases.

H. WORLEY KENDELL, ELLA V. FAY and BEATRICE WADE, American Occupational Therapy Association and University of Illinois College of Medicine, Department of Physical Medicine, Chicago:

Occupational Therapy: There will be actual demonstrations on patients, varying the patients from day to day. Included will be cerebral palsy, paraplegia, poliomyelitis, general medical cases and arthritis.

HOWARD A. RUSK and DONALD A. COVALT, New York University School of Medicine, New York, and MILDRED ELSON and CATHERINE WORTHINGTON, American Physical Therapy Association, New York:

Rehabilitation in Late Poliomyelitis: Methods and devices for the late rehabilitation of patients with poliomyelitis will be demonstrated with actual patients.

F. A. HELLEBRANDT, Baruch Center on Physical Medicine, Medical College of Virginia, Richmond:

Hydrotherapy: Hydrotherapeutic procedures of use to the general practitioner will be demonstrated.

A. RAY DAWSON, LOUIS B. NEWMAN, ROBERT BOYLE, EDWARD E. AVERY and A. B. C. KNUDSON, Veterans Administration, Washington, D. C.:

Physical Medicine and Rehabilitation in Tuberlosis and Thoracic Surgery: The ultimate goal in the rehabilitation of patients with tuberculosis and other diseases of the chest is not only physical recovery, but to train these individuals for future economic independence. The exhibit will include charts, photographs, slides, moving pictures and apparatus showing the various rehabilitation activities. Patients will also be demonstrated.

HENRY H. KESSLER, New Jersey Rehabilitation Clinic, Newark, N. J.:

Rehabilitation of Amputees: This exhibit will present methods of rehabilitating persons who have major amputations of upper or lower extremities. Proper use of the prosthesis will be demonstrated by patients.

The following demonstrators will assist in the presentation of the exhibit:

Frances Baker, San Francisco.
Harold Dinken, Denver, Colo.
Arthur C. Jones, Portland, Ore.
W. Jerome Lee, Richmond, Va.
Sedgwick Mead, St. Louis.
C. O. Molander, Chicago.
W. D. Paul, Iowa City.
Donald L. Rose, Kansas City, Kan.
Herman L. Rudolph, Reading, Penna.
Walter M. Solomon, Cleveland.
Augustus Thorndike, Boston.
George D. Wilson, Asheville, N. C.
Walter J. Zeiter, Cleveland.

Session on Physical Medicine at the Annual Meeting of the American Medical Association

During the annual meeting of the American Medical Association in Chicago there will be a session on physical medicine in the Section on Miscellaneous Topics. The session is scheduled for Thursday morning, June 24, 1948.

There was excellent attendance at the first session held in conjunction with the American Medical Association meeting in Atlantic City last June, and it is to be hoped that there will be an equally good attendance this year. Dr. George Morris Piersol has been appointed Chairman and Dr. Frank H. Krusen, Secretary of the session. See detail of program elsewhere this issue.

ARCHIVES of PHYSICAL MEDICINE

OFFICIAL PUBLICATION AMERICAN CONGRESS OF PHYSICAL MEDICINE

EDITORIALS

SYNERGISM OF ARTIFICIAL FEVER WITH DRUGS AND ANTIBIOTICS

There is considerable experimental and clinical evidence that a synergistic relationship exists between artificial fever and certain drugs and antibiotics. This may be of importance in some resistant cases of infectious disease which do not respond to the usual drug or antibiotic therapy. It is of importance in neurosyphilis, at least until it has been conclusively demonstrated that penicillin alone is curative. Since we are presenting in this issue a new combination of fever and drug therapy, a brief review of the subject seems in order.

In 1937 Ballenger, Elder and McDonald[1] were the first to present the combined use of sulfanilimide and fever therapy in a small series of ten cases of gonorrhea. They employed relatively small doses of sulfanilimide and mild fever therapy of 103 to 104 F. for three or four hours. All of their patients recovered with three such fever sessions given on alternate days, combined with 4 or 5 Gm. of sulfanilimide daily. Other investigators subsequently obtained almost as good results with combined fever and sulfonamide therapy in gonorrhea, but with higher fever and longer sessions.

White[2] in 1939 found, in vitro, that an increase in temperature from 37.0 to 39.0 C., caused a 100 fold increase in the streptococcocidal potency of sulfanilimide and sulfapyridine. In 1945 Lee and Foley[3] reported that, at a temperature of 37 C., 1.5 units of penicillin per milliliter of rabbit serum in vitro reduced the percentage of viable Staphylococcus aureus 75 per cent; at 39 C., 83 per cent; and at 42 C., 96 per cent. Likewise, Hoyt, Pratt and Levine[4] in the same year demonstrated in vitro a marked increase in the staphylococcocidal activity of penicillin by an increase of the incubating temperature from 37 C. to 41 C.

Eagle[5] in 1938 reported that the antispirocheticidal potencies in vitro of arsphenamine, neoarsphenamine, mapharsen, and bismuth were increased two fold by a 10 C. rise in temperature. In 1942 Boak, Carpenter and Warren[6] showed, in a well executed study, a definite synergistic relationship between fever therapy and neoarsphenamine in experimental syphilis in rab-

1. Ballenger, E. G.; Elder, O. F., and McDonald. H. P.: Sulfanilimide and Thermotherapy in Gonococcic Infections, J. A. M. A. 109:1037 (Sept. 25) 1937.

2. White, H. J.: Relationship Between Temperature and Streptococcocidal Activity of Sulfanilimide and Sulfapyridine, J. Bact. 38:549 (Nov.) 1939.

3. Lee, S. W., and Foley, E. J.: Effect of Temperature on Action of Penicillin in Vitro, Proc. Soc. Exper. Biol. & Med. 60:133 (Oct.) 1945.

4. Hoyt, R. E.; Pratt, O. B., and Levine, M. G.: Activity of Penicillin at Temperatures Above 37 C., J. Lab. & Clin. Med. 30:736 (Sept.) 1945.

5. Eagle, H.: The Minimal Effect Concentration of Arsenic and Bismuth Compounds on Treponema Pallidum in Vitro in Relation to Therapeutic Dose, Am. J. Syph. Gonor. & Ven. Dis. 23:310, 1939.

6. Boak, R. A.; Carpenter, C. M., and Warren, S. L.: Concurrent Treatment with Fever and Neoarsphenamine of Experimental Syphilis in Rabbits, Am. J. Syph. Gonor. & Ven. Dis. 26:291 (May) 1942.

bits. Fever alone cured 10 per cent of the animals, neoarsphenamine alone cured 33 per cent, and fever therapy combined with neoarsphenamine cured 75 to 100 per cent. The same phenomenon has been apparent, although to a much less marked degree, in the treatment of clinical neurosyphilis in the human.

In 1944 Eagle and Musselman[7] reported a marked increase in the spirocheticidal potency, in vitro, of penicillin with increasing temperature. Four different strains of spirochaetes were exposed for twenty-four hours to varying concentrations of penicillin at temperatures ranging from 8 C. to 40 C. At 8 C., 100 per cent of the organisms survived; at 22 C., 10 per cent; at 32 C., 1 per cent; at 36 per cent, 0.2 per cent; and at 40 C., only 0.02 per cent. These results were independent of the concentration of penicillin from 0.25 to 256 units per milliliter. In a recent study, Eagle, Magnusen and Fleishman[8] demonstrated that the potency of penicillin, in experimental syphilis in the rabbit, is increased eight to ten fold by a ten hour session of fever therapy at 104 to 105 F. They also pointed out that penicillin does not interfere and may be given simultaneously with any of the fever producing agents: malaria, typhoid vaccine, or cabinet fever.

In November, 1947, Curtis, Burns and Norton[9] presented 118 cases of neurosyphilis, in 75 of which penicillin alone was used in the treatment, while in 43 penicillin was combined with malarial inoculation. Of those treated with penicillin alone, 51 per cent showed clinical improvement, whereas 74 per cent of those given the combined treatment showed clinical improvement. The spinal fluid findings improved in 62 per cent of the penicillin treated cases and in 81 per cent of those in which the combined therapy was employed. The authors stated that the significance of these figures was still further enhanced by the fact that the combined treatment was used in the more severe cases.

Further evidence of the synergism existing between fever therapy and certain drugs is furnished by the report of Drewyer[10] appearing in this issue of the ARCHIVES. This author combined small doses of gold thiomalate with very mild fever therapy in the treatment of rheumatoid arthritis. The gold was administered once weekly and the fever therapy on alternate days. On the intervening days, other types of physical therapy were used. According to the author, 90 per cent of the patients with rheumatoid arthritis, 82 per cent of those with rheumotoid spondylitis, and 71.5 per cent of those suffering with both of these conditions showed improvement on this program of treatment. Furthermore, there were no toxic manifestations from the gold injections. With confirmation, this would appear to be a significant advance in the treatment of this difficult disease.

From the foregoing summary, it is evident that elevation of the temperature enhances the antibacterial activity in the body of the sulfonamides, the organic arsenicals, the bismuth salts, penicillin, and probably the gold compounds.

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7. Eagle, H., and Musselman, A. D.: Spirocheticidal Action of Penicillin in Vitro and Its Temperature Coefficient, J. Exper. Med. 80:493 (Dec.) 1944.

8. Eagle, H.; Magnusen, H. J., and Fleishman, R.: The Effect of Hyperpyrexia on the Therapeutic Efficacy of Penicillin in Experimental Syphilis, Am. J. Syph. Gonor. & Ven. Dis. 31:239 (May) 1947.

9. Curtis, C. C.; Burns, R. E., and Norton, D. H.: Neurosyphilis; Treatment with Penicillin Alone and with a Combination of Penicillin and Malaria, Am. J. Syph. Gonor. & Ven. Dis. 31:618 (Nov.) 1947.

10. Drewyer, G. E.: Low Grade Fever Therapy as an Adjunct in the Treatment of Certain Types of Arthritis, Arch. Phys. Med. 29:285 (May) 1948.

EFFECT OF ARTIFICIALLY INDUCED FEVER ON THE PERIPHERAL CIRCULATION

Wakim, Krusen and Elkins[1] present in this issue of the ARCHIVES an interesting study of the circulatory effects of artificially induced fever on the circulation of arthritic patients. Three methods of fever production were employed: the Kettering hypertherm, the hot tub bath and the intravenous injection of typhoid vaccine. Blood flow in the forearm was measured by the compensating plethysmokymograph.

Of the three methods, the Kettering hypertherm produced the greatest increase in the forearm volume, the hot tub bath the next greatest, and the typhoid vaccine the least. During the prodromal and chill phase following the intravenous injection of typhoid vaccine, the forearm volume decreased. This gave way to an increase in volume during the flush phase, but was always much less than that produced by the fever cabinet or the hot tub bath.

The average increase in blood flow was 152 per cent for the hot tub bath, 226 per cent for the cabinet and 51 per cent for the typhoid vaccine. The average percentage decrease in arm volume during the chill phase of the typhoid reaction was 53 per cent.

Such fundamental physiologic studies as this one point the way to the more rational use of therapeutic procedures. In this instance, it is apparent that if an increase in blood flow in the periphery is the objective of treatment, typhoid vaccine injection is not the method of choice. Another important consideration is that fever produced by physical means can be accurately dosed and easily controlled or discontinued as circumstances may demand. Although typhoid vaccine is economical and convenient to use, the response is more or less unpredictable and reactions may occasionally be serious.

1. Wakim, K. G.; Krusen, F. H.; Elkins, E. C.: Effect of Artificially Induced Fever on the Circulation in Arthritic Patients, Arch. Phys. Med. 29:274 (May) 1948.

PHYSIATRICY

Since the term "physiatrist" has become widely accepted to describe the specialist in physical medicine and since most persons believe it to be an entirely new word, it may come as a surprise to learn that in the Boston Medical and Surgical Journal for December 11, 1833, appeared an editorial entitled "Physiatricy." This simply confirms the old contention that there is "nothing new under the sun."

The editorial of 115 years ago pointed out that "the cardinal point on which everything in medicine, theory as well as practice, rests, is the relation and different reaction of the vital power, in connection with the different organization by which it acts, and the dead (chemical and mechanical) natural powers, subordinate to it." The editorial attributed to a Thuringian physician, Christian Wilhelm Hufeland (1762-1836), the introduction of the word "physiatricy" and mentioned that this was a "favorite term" of his. There was also reference to "the mode and principle of practice which the physiatricist would recommend." So the modern physiatrist may know that more than a century ago both the subject of physiatricy and the physiatricist were being discussed in the medical literature.

MEDICAL NEWS

American Board of Physical Medicine

The American Board of Physical Medicine, through its Secretary, Dr. Robert L. Bennett, announces that the dates and locations of the next examinations for certification to the Board will be as follows: Chicago, June 18 and June 19, 1948; Washington, D. C., September 4 and September 5, 1948.

Dr. Ewerhardt Retires

As of January 1, 1948, Frank H. Ewerhardt, M.D., who has had charge of the Department of Physical Therapy, Barnes Hospital and Washington University School of Medicine since 1915, retired and has been appointed Emeritus Chief of Physical Medicine to Barnes, McMillan, Maternity and St. Louis Children's Hospitals and Washington University Clinics. Dr. Ewerhardt will continue for the balance of the current year in teaching the present class of Physical Therapy students. He is also continuing his connection with the Veterans Administration Hospitals as Branch Consultant for the Ninth Area.

Dr. Sedgwick Mead, formerly associated with Massachusetts General Hospital, Boston, has been appointed Chief of Physical Medicine to Barnes, McMillan, Maternity and St. Louis Children's Hospitals and Washington University Clinics.

American Society of Physical Medicine

There were tentative plans being made for a meeting of the American Society of Physical Medicine just prior to the meeting of the American Medical Association in June. Since there will be a session on physical medicine during the meeting of the American Medical Association, and since arrangements are being made for the American Society of Physical Medicine to have a period for a longer business meeting and to conduct a scientific program at the meeting of the American Congress of Physical Medicine in September, it was decided to cancel plans for the meeting in June.

Southern Section of Congress Holds Session

The Southern Section of the American Congress of Physical Medicine met in Galveston, Texas, March 5. The program consisted of the following discussions: "The Occupational Therapist Looks at the Physiatrist," by Miss Mary Britton, Branch Occupational Therapist, Branch No. 10, Veterans Administration, Dallas; "Rehabilitation Problems in One VA Hospital," by Solomon Winokur, M.D., Chief, Physical Medicine Rehabilitation Service, VA Hospital, New Orleans; "Educational Trends," H. Worley Kendell, M.D., President, American Congress of Physical Medicine; "Hard Facts and Hard Cash," by Mr. George A. Walker, Executive Assistant, Physical Medicine Rehabilitation Division, Branch Office No. 10, Veterans Administration, Dallas, Texas, and "Functional Restitution in Functional Paralyses," a motion picture presented by Nathan H. Polmer, M.D., Branch Consultant in Physical Medicine to Branch No. 10 of the Veterans Administration.

Meeting of Pennsylvania Academy of Physical Medicine

A meeting of the Pennsylvania Academy of Physical Medicine was held Thursday, March 18, 1948, in Philadelphia, Penna. The following papers were presented and discussed. "Orthostatic Hypotension Following Hot Baths," by Dr. Steven Horvath; "Heating Human Tissue by Microwave Energy," by Dr. Ruth N. Miller and "Human Blood Pressure Response to Epiniphrine," by Dr. Allan De Wallis.

Residency Program at Hines

A three-year residency program in Physical Medicine has been approved by the Council on Medical Education, and hospitals of the American Medical Association, at the Veterans Administration Hospital, Hines, Illinois. The bed capacity at this hospital is approximately 3,500, consisting of general medical, general surgical, neuropsychiatric, and tuberculosis. The Physical Medicine Rehabilitation Service is a large, well-organized department, equipped with the latest apparatus for both diagnostic and therapeutic procedures. Residents with veteran status receives $3,300 per year. If interested, kindly write to K. A. Carroll, M.D., Manager, Veterans Administration Hospital, Hines, Illinois.

Dr. Bearzy Receives Appointment

Herman Bearzy, a member of the Congress has been appointed as official physician for the Olympic Teams.

Dr. House Appointed

Dr. Frederic B. House has recently been appointed Director of the Department of Physical Medicine of the Beyer Memorial Hospital, Ypsilanti, Mich. This is the first hospital to be established according to the new Michigan Public Act of 1945 whose purpose is to provide hospitalization for the community. Beyer Memorial Hospital is a general hospital of 155 beds serving western Wayne County and eastern Washtenaw County.

Dr. House is also Medical Director of the Department of Physical Medicine of St. Joseph Mercy Hospital, Ann Arbor, Mich.

Drs. Deaver and Watkins Speakers

Dr. George G. Deaver of New York City and Dr. Arthur L. Watkins of Boston presented a seminar in physical medicine as a part of the program of the thirty-third annual meeting of the American Association of Industrial Physicians and Surgeons, held in Boston, March 30 to April 2.

National Health Assembly

Pointing out that development of hospitals has not kept pace with the people's need for medical care or with medical advances, Oscar R. Ewing, Federal Security Administrator, has announced the formation of a section on hospital facilities to consider this aspect of the Nation's health problem at the National Health Assembly.

The Assembly, to be held in Washington, D. C., May 1-4, has been called by the Federal Security Administrator in response to the President's request of January 30 to Mr. Ewing for recommendations on feasible national health goals for the next ten years. Mr. Ewing has asked professional and lay leaders concerned with all aspects of health to advise him on framing his report to the President.

Dr. Charles F. Wilinsky, executive director of Beth Israel Hospital, Boston, Mass., and president-elect of the American Public Health Association, will be chairman of the hospital facilities section. He will be assisted in planning the work of the section by a small committee drawn from among delegates of the section.

In announcing the formation of the section on hospital facilities, Mr. Ewing said the present situation, nation-wide, with regard to hospital beds and services needs to be reviewed. He referred to the poor distribution of existing hospitals in the country as a whole and particularly to the scarcity of hospital facilities in rural areas. He suggested the desirability of an analysis of the roles of the various types of hospitals to determine their interrelationships. Facts developed on these and other points will be essential to a major consideration of the section — the Federal and State role with respect to the Hospital Survey and Construction Act of 1946.

In relation to its study of the hospital problem the hospital facilities section will also consider the nation's needs for health centers and diagnostic facilities.

The section on hospital facilities will be composed of about 40 persons. They will be professional people, lay experts and representatives of organizations having an interest in the hospital field. While the broad area of hospital problems will be outlined by Dr. Wilinsky and the committee, the specific agenda of the section will not be formulated until the first session of the section.

It was emphasized that advice from the "grass roots up" is being sought in the forthcoming "town meeting" on the nation's health needs. It was pointed out that spokesmen from the medical and health professions, for great national voluntary health organizations and for the citizens generally will have an opportunity to examine every facet of the problem.

While only one section of the National Health Assembly will be concerned specifically with the hospital problem, this topic cuts across most of the subjects to be considered by the 14 other sections. These include professional personnel, local public health units, chronic disease, maternal and child health, rural health, research, medical care, community planning, rehabilitation, dental health, mental health, nutrition, sanitation and industrial health.

Pamphlet on Orthopedic Nursing

The National League of Nursing Education, New York City announces a new pamphlet, No. 6, "Guide for an Advanced Clinical Course in Orthopedic Nursing," under the general heading "Courses in Clinical Nursing for Graduate Nurses." The central objective is stated to be the ability to utilize orthopedic principles in all nursing situations and to plan and carry out expert nursing for patients with orthopedic conditions. The cost is sixty cents.

Rehabilitation Program for Mexico City

The first Inter-American Conference on Rehabilitation of the Crippled and Disabled will be held in Mexico City, July 18 to 24, 1948, under the International Society for the Welfare of Cripples, and in cooperation with the Mexican Government. Those who may attend are official government delegates, members of the International Society, physicians, nurses, teachers, therapists and other professions, voluntary workers and anyone interested in the welfare of persons with disabilities.

Complex Simple Fracture

To the Editor:—Realizing that the term "simple fracture" as generally defined is thoroughly inadequate to describe the trauma received in many so-called simple fractures, I have added a new definition to the simple and compound fracture nomenclature, i. e., "complex simple fracture" (Key, J. A., and Conwell, H. E.: The Management of Fractures, Dislocations and Sprains, ed. 4, St. Louis, C. V. Mosby Company, 1946, pp 41-43).

A complex simple fracture is a fracture which is not compound but which has received severe trauma to either or all of the surrounding soft structures, i. e., skin, muscles, blood vessels and nerves. The bony fragments usually are displaced to a marked degree.

"Complex," as defined by "Webster's New International Dictionary," unabridged, is as follows: (1) an assemblage of related things; (2) a whole made up of complicated or interrelated parts.

The complex simple fracture should be further described as to the type of bony injury, for example: complex simple fracture (transverse, comminuted, spiral or oblique, as the case may be).

Through personal contacts and correspondence with the personnel of the three National Fracture Committees (American Medical Association, American College of Surgeons and American Academy of Orthopaedic Surgeons) with reference to their opinions and the necessity for such new fracture term or definition, there was unanimous agreement that

such a definition of complex simple fracture was a necessary one and should be generally accepted and used in its broad meaning.

I make an appeal that this new fracture definition or its relative equivalent be used by the members of the medical profession. Besides expressing more adequately the pathologic process in such type fracture it will mean improved records for the hospitals and convey a better description of the fracture to the insurance companies, compensation boards and other related agencies, thereby being of much help to the patient, the physician and every one concerned.

I will appreciate any opinion or suggestions relative to the foregoing.

H. Earle Conwell, M.D., Birmingham, Ala.

Corres., J. A. M. A. 135:531 (Oct. 25) 1947.

New Quarters for American Hearing Society

Beginning April 1, 1948, the American Hearing Society, which has been housed for twenty-five years in the Volta Bureau Building, 1537 35th Street, Northwest, will be occupying quarters at 817 14th Street, Northwest, Washington 5, D. C. Two factors influenced this move: an opportunity to secure a suite of offices (recently released by the Government) on the fifth floor, with elevator service, in a downtown location, and a decision by the Board of Trustees of the Volta Bureau to remodel their building.

Physical Therapists Nominated

Announcement is made by Major General Raymond W. Bliss, that the following physical therapists have been nominated for appointment in the Women's Medical Specialist Corps, Regular Army:

Beard, Genevieve	Captain
Burt, Josephine	1st Lt.
Ferrazzoli, Theresa M.	1st Lt.
Fowler, Elizabeth	1st Lt.
Jones, Nettie B.	1st Lt.
Miller, Jessie L.	Captain
Partridge, Miriam	1st Lt.
Reichert, Betty Lou	1st Lt.
Wenstrom, Hildegard V.	2nd Lt.
Westfall, Eleanor	Captain
Whitcomb, Beatrice	Captain
Whitehurst, Margaret	Captain

Clinical Fellowships in Physical Medicine

A limited number of clinical fellowships are available to physicians who wish to prepare for certification by the American Board of Physical Medicine.

Method of Administration. — Application may be made to the National Foundation at any time during the year. Selection of candidates will be made on a competitive basis by the Committee on Fellowships in Physical Medicine. This Committee is composed of members of the profession who are certified by the American Board of Physical Medicine. All decisions of the Committee regarding rejection or approval will be accepted as final by the National Foundation.

Tentative decisions regarding applications will be obtained from the Committee at periodic intervals, after all credentials have been assembled and a program of training has been submitted. However, final awards will not be made until candidates have been accepted for the first period of training.

Obligation of Those Accepting Fellowships. — In accepting a fellowship, each recipient must agree to practice as a specialist in physical medicine in the United States or its territories for a minimum of two years following completion of three years of clinical study. Both are required to qualify for the Board examination.

Eligibility Requirements. — 1. Graduation from a Class A school of medicine.

2. Completion of a rotating internship of not less than one year in a hospital approved by the Council on Medical Education and Hospitals of the American Medical Association.

3. License to practice medicine in one or more states.

4. Citizenship in the United States.

5. Age limit, 40.

6. Sound health as attested by a physical examination.

Financial Benefits. — Awards are based on the individual need of each applicant. Fellowships will cover a period of one, two or three years of clinical study, depending on the length of time required by the applicant to prepare for certification by the American Board of Physical Medicine.

Partial fellowships are available for qualified veterans to supplement the amount they will receive under the G. I. Bill of Rights.

General Information. — 1. Fellowships cover programs at training centers which have been approved by the Council on Medical Education and Hospitals of the American Medical Association for residencies in physical medicine. It is the responsibility of each applicant to arrange his own program, which must be planned to meet the requirements of the American Board of Physical Medicine. A list of the approved residencies may be obtained by writing to the Council on Medical Education and Hospitals, 535 North Dearborn Street, Chicago 10, Illinois.

2. Fellowships will be discontinued at any time for those fellows who do not continue to show satisfactory aptitude and ability. Reports will be obtained at regular intervals from the training center.

3. The National Foundation does not assume any responsibility for placing fellows in positions when training is completed.

4. Applications may be obtained from the National Foundation for Infantile Paralysis, 120 Broadway, New York 5, N. Y.

First International Poliomyelitis Conference

Ten world medical and research authorities on poliomyelitis have been named presiding officers for plenary sessions of the First International Poliomyelitis Conference to be held in New York City, July 12 to 17.

Hart E. Van Riper, M.D., medical director of the National Foundation for Infantile Paralysis, who is general chairman of the conference, also announced subjects for the sessions. He said these "were selected to give the most complete discussions possible on research on the disease and emthods of treatment approved by recognized medical standards."

Each of the presiding officers will be assisted by a panel composed of international authorities who have done especial work on particular phases of the problem. Papers on polio will be presented at each session and followed by open discussions.

Summaries of polio problems in other countries will be presented by official delegates who have been invited to represent their governments. Invitations have been extended through the State Department to more than 60 nations. In addition, some 20 other medical and scientific authorities from abroad will participate in the program.

Delegates to the conference will be divided into three categories including the official government delegates; institutional delegates representing invited universities, societies and scientific and philanthropic organizatons interested in research and treatment of virus diseases, and member delegates. The last group will include physicians and other scientific and professional persons qualified by recognized standards. A preliminary announcement bulletin outlining plans for the conference is being sent to recognized professional persons inviting them to attend as member delegates.

Dr. Van Riper noted that "work must be done on an international scale to reach higher degree of comparability of all problems presented by the disease, its treatment and research.

"Collection of local detailed epidemiological and immuniological studies from all parts of the world and correlation of such studies has never been accomplished on the necessary scale before," he said.

"It is planned that this conference will serve as a means of expediting distribution of vital knowledge on a worldwide basis.

"The first response from medical and scientific authorities in all countries who have been invited to participate in the program has been outstanding. Many in this country and abroad have declared that the problem has become so serious that poliomyelitis must be considered on an international basis with complete pooling now and in the future of all information available."

Plenary session presiding officers and subject follow:

Oswaldo P. Campos, M.D., clinical orthopedic surgeon, Hospital, Jesue, Rio de Janeiro, Brazil, "The Importance of Poliomyelitis as a World Problem."

Rustin McIntosh, M.D., professor of pediatrics, Columbia University, New York City, "Poliomyelitis; The Early Stage."

Arthur Steindler, M.D., professor of orthopedic surgery, State University of Iowa, Iowa City, Iowa, "Poliomyelitis: The Convalescent Stage."

Arvid Wallgren, M.D., professor of pediatrics, Royal Caroline Medical Institute, Stockholm, Sweden, "The Management of Poliomyelitis: The Convalescent Stage."

Carlos S. Ottolenghi, M.D., Oocente Libre de Orthopedia, Buenois Aires, Argentina, "The Management of Poliomyelitis: The Late Stage."

James E. Paullin, M.D., professor of clinical medicine, Emory University, Atlanta, Ga., "Bulbar Poliomyelitis."

Pierre L. LePine, M.D., director of laboratories, Pasteur Institute, Paris, France, "Immunology and Chemotherapy in Poliomyelitis."

Harry S. Mustard, M.D., Commissioner of Health, New York City, "The Public Health Aspects of Epidemic Poliomyelitis."

Thomas Parran, M.D., Surgeon General, United States Public Health Service, "Poliomyelitis Throughout the World." Official government delegates will present reports on poliomyelitis problems in their countries at this session.

The plenary sessions will be preceded by the opening meeting presided over by Irvin Abell, M.D., clinical professor emeritus of surgery at University of Louisville, Louisville, Ky. Delegates will be welcomed by Basil O'Connor, President of the National Foundation, and foreign delegates will be introduced by Morris Fishbein, M.D., editor of The Journal of The American Medical Association.

The conference, to be held in the Waldorf-Astoria Hotel, is being held under the auspices of the National Foundation for Infantile Paralysis with the cooperation of the following scientific societies and United States government agencies which have endorsed the program:

Department of State; office of the Surgeon General, Department of the Army; Bureau of Medicine and Surgery, Department of the Navy; United States Public Health Service, United States Children's Bureau, and Veterans Administration.

Also, American Academy of Orthopedic Surgeons, American Academy of Pediatrics, American Association of Pathologists and Bacteriologists, American College of Physicians, American College of Surgeons, American Congress of Physical Medicine, American Heart Association, The American Medical Association, American Pediatric Society, American Physiological Society, American Physiotherapy Association, American Public Health Association, Association of Military Surgeons of the United States, Federation of American Societies for Experimental Biology, National Research Council, National Society for Crippled Children and Adults and Western Surgical Association.

Among the other distinguished professional authorities from abroad who will participate in the program are. H. J. Seddon, M.D., medical director, Wingfield-Morris Orthopedic Hospital, Oxford, England; J. H. Gear, M.D., South African Institute for Medical Research; Johannesburg, South Africa; Herman Romero, M.D., professor of health, University of Chile, Santiago, Chile, and S. van Creveld, M.D., professor of pediatrics, University of Amsterdam, Amsterdam, The Netherlands.

Also, Fritz Buchtal, M.D., Universitetets, Neuroysiologiski Institut, Copenhagen, Denmark; Juan Farill, M.D., acting president, International Society for the Welfare of Cripples, Mexico City, Mexico; Alfonso Montague, M.D., Lima, Peru; Sven Gard, M.D., professor of bacteriology, Statens Bakteriologiska Lagoratorium, Stockholm, Sweden; G. Paredes, minister of public health, Republic of Panama, and L. N. Silverthorne, M.D., Hospital for Sick Children, Toronto, Canada.

Aid for "Forgotten Children" Is Proposed by New York State Group

After two years of intensive study that has included numerous hearings all over the state, visits to clinics and consultations with leading specialists, a Joint Legislative Committee to Study the Problems of Cerebral Palsy recently has filed its report with the New York Legislature. The report calls for a long-range program to provide medical care, rehabilitation and special education for a group the committee has rightly termed "the most neglected group of handicapped children in the state."—Rusk, N. Y. *Times*.

Award Dinner in Honor of General Hawley

The Waldorf-Astoria was the scene March 19, of an award dinner in honor of General Paul R. Hawley, held by the National Council on Rehabilitation. President Henry H. Kessler, made the award. Among the speakers were Major General R. W. Bliss, Howard A. Rusk and Carl R. Gray, Jr.

New Cerebral Palsy Booklet

Latest medical knowledge on the causes and treatment of cerebral palsy is contained in "The Problem of Cerebral Palsy Today," a new 57-page booklet written by Dr. Meyer A. Perlstein.

The contents include a description of the problem of the cerebral palsied child, a suggested state program, professional training, research needs and cerebral palsy as a socio-economic problem. Of special interest are sections devoted to an expanded diagrammatic chart of how a model cerebral palsy program operates and some hypothetical case histories illustrating the mechanics of the cerebral palsp program.

Portrait photographs of young cerebral palsy victims, and an non-technical style of writing make "The Problem of Cerebral Palsy Today" interesting and informative reading.

Copies of the booklet can be obtained from the National Society for Crippled Children and Adults, Inc., 11 S. La Salle Street, Chicago 3, Illinois, at 25 cents each.

Army Nurses Study Reserve Recruiting Problems

Fifty Army Nurse Corps leaders from continental United States and overseas commands have been summoned by The Surgeon General to Washington for a two-day conference to discuss the new Army Nurse Corps Section of the Officers' Reserve Corps and to map out a program to enroll 29,000 Army Nurse Corps Reserve officers during 1948.

The Army Nurses attending the conference are chief nurses of the six Army areas in the U. S. and of Army General Hospitals, and the directors of nursing services in the Far East Command, in Europe, and the Antilles.

VA Appointments

All members of the Veterans Administration national advisory group on medical problems have accepted reappointment by Carl R. Gray, Jr., Administrator of Veterans Affairs.

Chairman of the group is Dr. Charles W. Mayo, of the Mayo Clinic, Rochester, Minn. Other members are: Dr. Charles F. McCuskey, Glendale, Calif.; Dr. John S. Voyles, St. Louis, Mo.; Dr. Paul Titus, Pittsburgh, Pa ; Dr. Guy W. Brugler, Boston, Mass. Dr. W. S. Middleton, Madison, Wis.; Dr. Francis J. Braceland, Rochester, Minn.; Dr. Claude C. Coleman, Richmond, Va.; Dr. Francis M. McKeever, Los Angeles, Calif.; Miss Katherine J. Densford, Minneapolis, Minn.; Dr. Derrick T. Vail, Chicago, Ill.; Dr. A. R. Shands, Jr., Wilmington, Del.; Dr. Dean M. Lierle, Iowa City, Ia.; Dr. Roy Kracke, Birmingham, Ala. Dr. A. C. Christie, Washington, D. C.; Miss Eleanor Cockerill, Pittsburgh, Pa.; Dr. J. Stewart Redman, Philadelphia, Pa.; Dr. George F. Cahill, New York and Dr. William A. Hunt, of Chicago.

Meeting of the American Academy for Cerebral Palsy

The American Academy for Cerebral Palsy will hold its first annual meeting in Baltimore, on May 30 and 31, 1948.

BOOK REVIEWS

DISABILITY EVALUATION. PRINCIPLES OF TREATMENT OF COMPENSABLE INJURIES. By *Earl D. McBride,* B.S., M.D., F.A.C.S. Diplomate American Board Orthopedic Surgery. Assistant Professor in Orthopedic Surgery, University of Oklahoma School of Medicine; Attending Orthopedic Surgeon to St. Anthony's Hospital; Assistant Orthopedic Surgeon to Wesley Hospital; Visiting Surgeon to W. J. Bryan School for Crippled Children; Chief of Staff to Bone and Joint Hospital, Oklahoma City, Okla. Fourth Edition, revised. Cloth. Price, $12.00. Pp. 642, with 400 illustrations. Philadelphia, London, Montreal: J. B. Lippincott Co., 1948.

This is a book on the function of the body and its parts and the functional loss that occurs following various injuries. Each injury is discussed as to diagnosis, treatment, the mechanical and functional alterations and the author's system of arriving at a percentage basis of disability. The latter feature is unique. He uses what he terms his "measuring rod" for the numerical basis of the evaluation of disability. This has a definite practical value. The author provides a percentage for such factors as "delayed action, awkwardness, weakness, insecurity, fatigue, lowered safety factor and impaired prestige. Numerous tables are given to demonstrate how this measuring rod is applied to various extremities, the eye or the ear.

In the first chapter consideration is given to such subjects as workmen's compensation laws, a basic survey of disabilities, an explanation of legal terminology, sensible advice to the physician as an expert witness and other helpful suggestions as to records and reports.

A 38 page insert is included which gives the author's composite schedule of evaluation for partial permanent disability representing a composite of approximate ratings and serving as a means of standardization and contains "the sequences of past experiences in respect to medical opinion as well as the precedence of established acts of legal significance." Anyone concerned with equitable compensation could follow such a method with satisfaction. The author has an appreciation of the problems of patients with industrial injuries. He realizes that "it is a trait of human nature to exaggerate or to make false claims for the purpose of self aggrandizement," but at the same time "sure malingering or fraud is not frequent." He does not consider malingering impossible for he does include a few suggestions to detect malingering but the spirit of the book is exemplified in the statement in the preface that "organized society has assumed the responsibility by providing monetary compensation and social rehabilitation."

Chapters on ankylosis consider the various joints involved. Descriptions and illustrations of normal motion are followed by a discussion of ankylosis, with sensible consideration of indications and contraindications of treatment. The disabilities that are encountered with joints are described in different positions not only from the viewpoint of its difficulties but also from that of its readjustment possibilities which should be of particular concern to persons interested in physical medicine. Many practical applications are suggested.

In the sections on fractures, a few pages discuss the problem of physics in fractures which explain how certain forces produce rest, equilibrium and motion. The fractures of different bones are individually considered with brief descriptions of the sites of fractures and their treatment and a more thorough discussion of disabilities following such fractures both as to permanent and partial disability. Each chapter concludes with "readjustment possibilities," which alone justifies the book. Chapters on the industrial back, the trunk, injuries to the eye and ear, amputations, nerve injuries, head injuries are presented in the same plan as for disabilities involving other structures.

The last chapter is entitled "the employment of the disabled person" and concludes with 22 pages which list the different types of work with code numbers representing the various parts of the body that may be disabled but still permit the person to work at the designated job.

This volume should be indispensable to physicians in industrial work and to orthopedic surgeons. It should be of much value to physiatrists who are often consulted about the disability of patients. It should be particularly stimulating to anyone working in a department of physical medicine not only because it presents the disabilities from a functional standpoint but also gives suggestions for possibilities of readjustment. The book, too, can be highly recommended to teachers in schools of physical and occupational therapy since kinesiology is interpreted clearly and practically in terms of working ability.

THE PERIPHERAL CIRCULATION IN HEALTH AND DISEASE. By *Robert L. Richards,* M.D., Rockefeller Fellow in Medicine; formerly Assistant Physician, Neurovascular Unit, Gogarburn Hospital, Edinburgh. With a foreword by *J. R. Learmonth,* C.B.C., Ch.M., F.R.C.S.E. Regius Professor of Clinical Surgery, and Professor of Surgery in the University of Edinburgh. First edition. Cloth. Price, $6.00. Pp. 153, with illustrations. Baltimore: The Williams & Wilkins Company, 1946.

In this short but carefully prepared monograph, the author gives an excellent, concise discussion on the anatomy and physiology of the vasomotor system. He explains both nervous and humoral

control of blood vessels. The sympathetic innervation of blood vessels of the extremities is given in good detail, including the four neurons from the cortex to the endings of the postganglionic fibers in the wall of the blood vessel. A study of the influence of environmental temperature, alterations in metabolism, changes in posture and local factors, on vasomotor activity is presented.

The author found induced vasodilatation by "limb immersion" a simple and satisfactory method of testing the capacity of the peripheral vessels to dilate. He showed that this reflex vasodilatation is the result of both afferent nervous impulses arising in receptors in the immersed limbs and a rise in blood temperature. The value of diagnostic nerve block also is considered. Occlusive vascular disease, the Raynaud phenomenon, and injuries to peripheral nerves are discussed, including diagnostic and prognostic procedures and the effects of sympathectomy. He presents evidence supporting the hypothesis that the Raynaud phenomenon is the result of "local fault" in the digital arteries, which may be related to the innervation of the arteries.

His observations on injuries to peripheral nerves indicate that the vasomotor state of the denervated cutaneous areas passes through two phases: (1) an initial phase of vasodilatation (warm phase), and (2) a second permanent phase (the cold phase), in which the temperature of the area approximates that of its environment.

The immersion-foot syndrome is considered in detail with its clinical manifestations, including a description of the prehyperemic, hyperemic and posthyperemic phases.

DISEASES OF THE JOINTS AND RHEUMATISM. By *Kenneth Stone,* D.M. (Oxon.), M.R.C.P., Honorary Physician, B.R.C.S. Clinic for Rheumatism, Peto Place, London, Physician B.R.C.S. Clinic for Rheumatism and Physical Treatment, Kensington, formerly Senior Demonstrator of Pathology and Rose Research Fellow, St. Bartholomew's Hospital. Cloth. Price, $6.50. Pp. 362. New York, New York. Grune & Stratton, 1947.

This book is proposed for "students . . . in the 'rheumatic diseases'" and the subjects are approached in a "mood of scientific criticism." Actually, the presentation is opened and closed on a philosophic plane, without benefit of the scholarly academic approach to which students of rheumatic disease are entitled. There is merit in the devoting of chapters to individual joints as well as to various arthritic diseases, but the outline and organization of this work are sketchy and incomplete. Sprain of the ankle joint and treatment of such a sprain are described in the chapter on rest and movement (p. 44), although painful ankles and feet constitute the subject of a separate chapter. Olecranon bursitis is not mentioned in the chapter on the elbow. The subcutaneous nodule of rheumatoid arthritis is described in a subheading under rheumatic fever. Intermittent hydrarthrosis is described as a type of psoriatic arthritis (p. 130), whereas in a subsequent chapter on painful knees, this articular condition is discussed without mention of a proposed relationship to psoriasis (pp. 255-257). Brachial neuralgia (p. 355) is considered in the chapter on painful disorders of the back, but is not described in a chapter on the painful shoulders (pp. 190-206). No mention is made of roentgen therapy for subacromial bursitis, or any other lesion of the shoulder, although this treatment is considered for osteo-arthritis.

Rheumatoid arthritis and its treatment, one of the most formidable and most difficult problems confronting the rheumatologist, is presented in a chapter 13 pages in length. In other chapters, only 2 lines are devoted to the treatment of osteo-arthritis of the spine (p. 168), and the action of muscles (which the author considers "important") is discussed in less than 1 page (p. 34). By comparison, 12 pages are devoted to the classification of rheumatic disease.

Competent American rheumatologists cannot fail to take exception to such statements as: "The gonococcal complement fixation test, carried out on either joint fluid or blood, is of far greater diagnostic value [than search for the gonococcus]" (p. 42); "These two [tuberculous arthritis and syphilitic arthritis] are the only types of arthritis in man known with certainty to be due to invasion of the joint tissues by micro-organisms" (p. 89); "overeating is a comparatively rare cause of obesity" (p. 244); the "diagnosis [of osteo-arthritis of the spinal column] is in the hands of the radiologist . . ." (p. 166).

More clarification of English usage of terms such as "chronic infective arthritis" and "arthritis of the rheumatoid type" as distinguished from rheumatoid arthritis would be helpful, at least for American students of rheumatic disease. Palindromic rheumatism is mentioned only as a brief footnote in the discussion of intermittent hydrarthrosis (p. 256). Subfascial herniation of fat (Copeman, 1944) is completely omitted. Tuberculous arthritis is greatly overemphasized throughout the book, whereas the subject of pain in the lower part of the back suffers from oversimplification. There is misdirected emphasis on "the conditions of work in a modern industrial city" and "colonic stasis and spasm" as factors involved in muscular rheumatism.

Those interested in physical medicine will find in this volume a heavy emphasis on electrotherapy of various types for nearly all the varieties of arthritis discussed. Use of the contrast bath is described in one brief sentence, while 8 sentences are devoted to the faradic foot bath (pp. 58 and 60). Galvanism is proposed for the absorption of deposits of urate in tophaceous gout (p. 147).

References are conveniently located as page footnotes, but most of them are bibliographically incomplete. The entire book contains less than 150 references; of these the most recent are 7 from 1944 to 1946. In the chapter on gout only 2 references are given — one printed in 1868 and one in 1920. The type is clearly readable and the text is notably free of typographical errors.

The author is to be commended for his slight attention to the use of "vaccines" for rheumatoid arthritis, for deploring the lack of precise usage

of the term "fibrositis" and for deprecation of the "fibrositic nodule."

Some theoretic aspects of psychogenic rheumatism ("the common rheumatism," "rheumatic myalgia") are presented in part 2 (97 pages). The author's suppositions are summarized in the statement that "rheumatic myalgia is a disorder of skeletal muscle secondary to a functional vascular disturbance" (p. 292). However, after considerable discussion, the reader is told that the treatment for muscular rheumatism is the same whether or not "little thought is given to the disturbances of the vegetative nervous system" (p. 295).

Although this book may interest some students of rheumatic disease, its deficiencies from the point of view of most American rheumatologists largely detract from its usefulness in the United States unless the reader is desirous of a superficial introduction to another author's (English) concepts of rheumatic disease.

DYING, APPARENT-DEATH AND RESUSCITATION. By *S. Jellinek,* M.D., Professor of Electro-pathology at the University of Vienna; Lauréat de l'Institut de France. Cloth. Pp. 263. Price, $3.50. Baltimore: The Williams & Wilkins Company, 1947.

This book represents the result of a medical lifetime of collecting data on the process of dying. The author has remarkable enthusiasm for the subject but very little scientific data for a book of this size. There are frequent repetitions and other defects of style so that the book is not recommended as a textbook for students and practitioners of medicine as the author primarily intends it.

A TEXTBOOK OF CLINICAL NEUROLOGY. WITH AN INTRODUCTION TO THE HISTORY OF NEUROLOGY. By *Israel S. Wechsler,* M. D., Clinical Professor of Neurology, Columbia University, New York; Neurologist, Mount Sinai Hospital; Consulting Neurologist, Montefiore Hospital and Rickland State Hospital, New York. Sixth Edition. Cloth. Price, $8.50. Pp. 781, with 162 illustrations. Philadelphia and London: W. B. Saunders Co., 1947.

This is the sixth edition of a work which was originally published in 1927. It is rightfully considered a classic and its popularity has been justly earned. This edition brings the book up to date and includes the progress that has occurred in the field of neurology during the past few years.

Minor criticism might be directed at a few statements such as "curare may favorably affect the paralysis" of poliomyelitis and "generally it is wiser to sacrifice the life of the fetus than that of the mother" in the treatment of chorea. The treatment of poliomyelitis is covered in one page which would be disappointing to anyone interested in physical medicine but this is understandable in a book covering the entire field of neurology. Herniation of the nucleus pulposus is considered only on the differential diagnosis of two other disorders which is not in keeping with its popularity at the present time.

The book can be recommended as one of the best single volumes for both students and clinicians who wish a clear and concise presentation of the subject.

THE TREATMENT OF RHEUMATISM IN GENERAL PRACTICE. By *W. S. C. Copeman,* O.B.E., M.A., M.D. (Cantab.), F.R.C.P. (London). Physician in Charge, Department of Chronic Rheumatic Diseases, West London Hospital; Physician, B.R.C.S. Clinic for Rheumatism, Peto Place, Hospital of St. John and Elizabeth and Cheyne Hospital for Children; Chairman of the Chartered Society of Physiotherapy and Medical Secretary, Empire Rheumatism Council. Fourth Edition. Pp. 258. Paper. Price, $4.00. Baltimore: The Williams & Wilkins Co., 1946.

This work first appeared in 1933. This edition attempts to bring the material up to date. The first part of the book gives a general discussion of the common types of arthritis: rheumatic fever, rheumatoid arthritis, osteoarthritis, gout, spondylitis and since it is written by an Englishman naturally a chapter on fibrositis. The chapters aim to give a brief sketch of these various forms and specific remarks for their treatment. The second half considers "Treatment" with chapters devoted to fecal sepsis, medicinal, diet, vaccines, physical methods, endocrines, spas, etc. This latter part of the book repeats and enlarges upon the remarks which are included in the section on the specific types of arthritis.

Many clinicians would disagree with numerous statements found in the book; for example his classification of rheumatoid arthritis into (1) due to infection and (2) not due to detectable infection with the statement "where a causative focus of infection is found should, strictly speaking, no longer be termed rheumatoid, but toxic or infective arthritis" would call for much violent disapproval. It seems strange that he believes the word atrophic arthritis is a "good name" which was the term originally used in America until the Americans adopted the British word "rheumatoid." Is Dr. Copeman hoping the British will adopt the American term? In the chapter on gout he expresses his opinion on foci of infection which would be frowned on by most rheumatologists and is best shown by this quotation "no devitalized tooth should be allowed to remain, even though it is performing some useful mechanical task such as anchoring a bridge or plate, since such teeth are invariably infected at their roots." The medications recommended for gout leave much to be desired; several drugs suggested are not generally used today and the one drug colchicine, which is considered the most effective by many rheumatologists is incorrectly prescribed.

In the treatment of chorea no mention is made of fever therapy, this may be an oversight. It is definitely indicated and if not by physical means certainly by the use of foreign proteins or bacteria. Although most Americans would applaud his cynical definition of fibrositis as "any pain of which the medical men were unable to diagnose the cause," it is questionable if his treatment would be appreciated by many patients. He recommends "heating a platinum loop to red hot over a spirit lamp by the bed side and firmly touch the skin over the affected area in several places a few inches apart" or "an electric cautery" or "a strong ery-

thema dose of ultraviolet should impress any patient favorably."

The emphasis placed on physical medicine is certainly commendable and his quotation "no scheme of treatment for chronic arthritis can be considered complete unless physical methods under skilled direction are provided" is borne out by devoting 86 pages in the second part of the book to various types of physical medicine and by considering physical methods throughout the earlier chapters. Not all will agree with the importance of mud therapy, colonic therapy and several forms of hydrotherapy that are included and the statement that "non luminous heat rays have a greater skin penetration than have the radiant heat rays" is obviously wrong — however this section of the book is probably the best and the most acceptable.

The book is a general discussion of the more common forms of chronic arthritis presented in a rather pleasant and optimistic manner but should have criticism leveled at its inclusion of controversial material and dubious therapy. The author is to be credited for his respect and understanding of physical medicine.

MUSCLE TESTING: TECHNIQUES OF MANUAL EXAMINATION. By *Lucille Daniels*, M.A., Director and Associate Professor of Physical Therapy, Stanford University; *Marian Williams*, M.A., Assistant Professor of Physical Therapy, Stanford University, and *Catherine Worthingham*, M.A., Director of Professional Education, The National Foundation for Infantile Paralysis, Inc. First edition. Paper. Pp. 189, with 349 illustrations. Philadelphia and London: W. B. Saunders Company, 1946.

This is a paper-bound booklet, illustrated by line drawings and written in outline form. In the introduction the authors have given a brief discussion of the history and development of manual strength-testing of muscles, an outline in which the different methods of grading of muscle-testing are compared, and an outline of the sequence of positions of the patient used to avoid frequent turning during muscle-testing examination.

The remainder of the book is arranged in outline form. The strength test for the muscles is given according to motion. Muscles which are prime movers are listed first; the nerve supply, origin and insertion are given. Along with this is a listing of the accessory muscles involved in the motion to be tested. The muscles which are prime movers are illustrated by clever line drawings. Opposite these are line drawings which show the position in which the patient should be placed for the various tests and according to the various gradings of the muscle strength, such as "normal," "good," "fair" and "poor" strength, "trace," or "zero." The various test positions and descriptions of rating of strength shown are more or less standard, and are those used by almost all workers doing muscle-strength testing in poliomyelitis.

The contents of the book are cleverly arranged and well illustrated. The material presented can be used as a quick review of muscle function, as well as testing. The authors' classification of the muscles and their description of some of the functions perhaps could be questioned in some respects, such as the statement that the tensor fascia is the prime mover in abduction of the hip. However, in general, the book is well prepared and can be recommended highly to students of physical therapy, to therapists interested in muscle re-education and muscle-testing, and as a review for physicians.

WOMEN IN INDUSTRY: Their Health and Efficiency. Issued Under the Auspices of the Division of Medical Sciences and the Division of Engineering and Industrial Research of the National Research Council. Prepared in the Army Industrial Hygiene Laboratory. By *Anna M. Baetjer*, Sc.D., Assistant Professor of Physiological Hygiene, School of Hygiene and Public Health, The Johns Hopkins University. Cloth. Pp. 344. Price, $4.00. Philadelphia: W. B. Saunders Company, 1946.

This book makes available information for the proper placement of women in industry when their service is required. In the first section data is presented in relation to physique, fatigue and physical stress, together with types and arrangements of work and the personnel policies and employment. In the second section sick absenteeism is discussed; and in the third and fourth, accidental injuries and occupational diseases. In the final section gynecological and obstetrical problems are elaborated upon. This is a valuable contribution to the field of occupational medicine and should be available to all industrial physicians and to personnel departments.

MODERN TREATMENT OF PEPTIC ULCER. By *Asher Winkelstein*, M.D., B.S., Associate Physician for Gastroenterology and Physician-in-Charge of The Gastrointestinal Clinic, The Mount Sinai Hospital, New York; Assistant Professor of Clinical Medicine (Gastroenterology), Columbia University, New York City. Cloth. Price, $4.00. Pp. 193 with 26 illustrations. New York: Oxford University Press, 1948.

This monograph is a concise critical analysis of the more recent advances in the modern treatment of peptic ulcer, and a brief review of the etiology, pathology, and pathologic physiology, chiefly as it affects the rationale of ulcer therapy. The methods of treatment taken into consideration are the intragastric drip therapy, vagotomy, the psychosomatic approach, the protein hyperelimination therapy and enterogastrone therapy. Continuous intragastric drip therapy is discussed in considerable detail to emphasize its present-day importance in the medical managment of refractory cases. This book is recommended to physicians interested in the clinical management of the ulcer problem.

PHYSICAL MEDICINE ABSTRACTS

Surgical Treatment of the Peripheral Nerve Injuries in General Hospitals. Francis C. Grant, and Eugene Spitz.

Pennsylvania M. J. **51**:521 (Feb.) 1948.

A review of peripheral nerve injuries seemed indicated when an examination of records showed that in roughly 60 per cent of the cases the fact that peripheral nerve trauma was present had been completely overlooked by the physician who first cared for the patient. The surgical technic is described. The types of injuries included 27 patients with an injury to the ulnar nerve, 31 patients with median nerve injury, 11 with injury of the radial nerve, and five patients, sciatic involvement.

It is pointed out that the surgeon's duty toward a patient suffering from a nerve section does not stop after nerve suture. The problem of recovery is not only to restore the conductive neural mechanism but to maintain the function of the muscles on which the nerve impulse will ultimately act. Passive movement of paralyzed fingers and active movement of adjacent normal muscles must be insisted upon immediately after suture. Any splinting necessary to maintain the limb in an abnormal position to prevent undue tension on the suture line must always be planned so that the fingers and other adjacent joints may be moved.

If only one of the nerves to an extremity is involved, the problem will be relatively simple, since the normal movement of adjacent muscles will go far to keep the paralyzed groups in trim. But when two of the three nerves are injured, then physical therapy, passive movement and electrostimulation are especially important and must be carried out promptly and for long periods. No matter how perfect the technic of nerve suture may be, if the muscles to be reenervated are allowed to become atrophied for fibrotic, the end result will be unsatisfactory. Cooperation on the part of the patient is, therefore, essential, for he must use his limb to the best of his ability at all times and willingly submit to physical therapy for lengthy periods, if the maximum in recovery of function is to be obtained.

Reactions to Cold Weather.

Editorial, Lancet **254**:329 (Feb. 28) 1948.

The body protects itself against chilling by increasing heat-production and reducing heat-loss. In "temperate" climates people can be divided roughly into two classes, an active and a passive, according to which of these two methods they favor in cold spells. The active class are braced by the cold; they speak sincerely of "this healthy weather," meaning that it stimulates them bodily and mentally and brings them a sense of well-being. Conversely, the passive class are incapacitated and made miserable by the cold; they huddle themselves in rugs and thick clothes and are chilled and cyanosed for hours after a cold immersion.

The few unfortunates subject to true cold-sensitivity have merely to immerse one hand in cold water to produce a general urticaria or angioneurotic edema. In others peripheral chilling produces a paroxysmal haemoglobinuria; if a sample of their blood is cooled to 5 C. and then rewarmed to body-heat hemolysis occurs through an autohemolysin produced by the cold. Heightened muscular tension may be important in the causation of "muscular rheumatism," especially where a localized icy draught rapidly chills warm muscles. But it is the tendency of cold to precipitate reflex spasm of vascular and visceral plain muscle that is the most potent cause of trouble.

Brachial Neuralgia. W. Russell Brain.

Lancet **254**:393 (Mar. 13) 1948.

Much work has been done recently on those neurovascular disorders that cluster round the root of the neck, a region that the course of human evolution has left in a plastic state and therefore liable to variation. As regards treatment, so long as the symptoms and signs are purely sensory, the condition may safely be treated by exercises of the shoulder-shrugging type; but, when muscular wasting or vasomotor symptoms are present, surgery is essential and the operation should be fully exploratory.

Electronarcosis. Its Value and Its Dangers. Gerald Garmany, and Donal F. Early.

Lancet **254**:444 (Mar. 20) 1948.

Three types of complications were encountered. A sharp hemoptysis developed in 1 patient and treatment was promptly stopped. The second complication was severe clouding of consciousness, which in 2 patients, aged 38 and 44 years, lasted for many days; there was no indication in either of these patient cases of preceding cerebral arteriopathy. The third, and most important, complication was collapse. This occurred in 7 patients, and in only 2 of these was the treatment in face of the risk, despite the anxiety to give the therapy a reasonable test.

The results of treatment with electronarcosis in 28 cases, including 21 of schizophrenia, are outlined. In no case of schizophrenia was any lasting improvement obtained; and in all but 2 not even transitory improvement was obtained. The authors find little evidence that electronarcosis has contributed to the physical therapy of the psychoses. The dangers inherent in the treatment appear to be considerable.

Tonoscillography After Exercise in Peripheral Vascular Disease and Coarctation of the Aorta. B. Ejrup.

Am. Heart J. 35:41 (Jan.) 1947.

Exercise-Tonoscillographic studies have been carried out in normal subjects, in patients with peripheral circulatory disturbances and in patients with coarctation of the aorta. The peripheral circulatory disturbances were divided into three groups. (1) demonstrable organic arterial circulatory disturbances; (2) intermittent claudication; and (3) miscellaneous types.

A method for automatic oscillographic blood pressure recording is described. By this method oscillograms can be taken in succession without any venous congestion in the examined limb. The oscillograms are written vertically on an endless paper perpendicular to the length of the roll so that variations of the blood pressure and pulsations can be observed easily. The apparatus is especially suited for the taking of oscillograms after exercise when several successive oscillograms are required to record the type and length of the postexercise period.

The procedure involved in exercise-oscillography is described. Oscillograms are first taken with the patient at rest in the horizontal position. Work tests are then carried out on the Nylin stairs or an ergometer bicycle, after which the oscillograms are repeated in the horizontal position during the postexercise period. In normal cases an increase of blood pressure and pulsations is observed after exercise, whereas in cases in which organic vascular changes have been demonstrated and in cases of typical intermittent claudication, an inverse reaction with decrease of blood pressure and lowering of the pulsations is obtained in the lower limbs.

In cases of coarctation of the aorta, a pathologic reaction occurs with no increase of blood pressure. In some cases the pulsations are diminished while in others they may be slightly improved. This pathologic reaction disappears after operation and the oscillographic picture at rest and after exercise becomes normal.

Cases of vague symptoms arising in the legs which do not constitute true intermittent claudication show, with few exceptions, a normal oscillogram after exercise.

The pathogenesis of the abnormal exercise-tonoscillograms is discussed and the value of exercise-tonoscillography in the early diagnosis of organic peripheral arterial circulatory disturbances is pointed out. Its value and importance in the differential diagnosis between structural and functional changes of the arteries are considered.

Ninth Day Erythema Showing Photosensitivity. Chaim Berlin.

Arch. Dermat. & Syphil. 56:771 (Dec.) 11947.

As to the special localization in the two cases, photosensitivity to arsenicals and also to arsphenamine has occasionally been observed, but, as far as known by the author, not of the type of ninth day erythema. On the other hand, these patients were not exposed to the sunlight during the treatment. In case 1 the disease occurred in November, but the patient's skin had been exposed to the sun and was considerably tanned after lengthy service in the Western Desert. The second patient had fair skin, and her exposed areas were rather tanned after a twenty-five year stay in this country. In the classic cases of photosensitivity it is believed that the characteristic cutaneous changes are frequently not due to direct irradiation by the sun. The skin which has been altered by the sun and light possesses a lower resistance and is more susceptible to sensitization. The damaged skin plays some role in the localization of the common arsphenamine dermatitis (Silberstein, Keim) but no such relation is so far known in the disease under consideration. Nevertheless, the impression is that in the cases herein reported damage to the skin by the sun might be in a great degree responsible for the special localization of erythema.

Classification of Peripheral-Nerve Injuries. Robert Roaf.

Lancet 254:242 (Feb. 14) 1948.

The concept of axonotmesis may be dangerous, in so far as the term is often applied to cover a multiplicity of states and lesions in which spontaneous recovery is by no means certain. This may lead either to unnecessary delay in surgical treatment or to neglect of other therapeutic measures. There are grounds for believing that most cases diagnosed as degenerative lesions in continuity are either nondegenerative lesions in which the delay in recovery is due to other factors, or partial lesions in which a varying proportion of fibres have been permanently damaged. If this proportion exceeds 50 per cent it is usually wise to perform a resection and suture.

A classification must be helpful as well as accurate; a single nerve-fibre is at any one moment either capable or incapable of conducting impulses. If incapable, it is in one of three conditions: (1) able to recover spontaneously without interference; (2) unable to recover spontaneously owing to solution of continuity and intervening scar tissue; or (3) subject to external pressure. (If the compressing agent is removed early, the nerve-fibre is usually able to recover, but at a later stage it may have suffered irreparable damage.)

In a nerve trunk, which is the unit with which the clinician deals, there are many different fibers, and their resistance to trauma varies. Therefore innumerable different syndromas may develop. According to the author nerve injuries fall into four classes: (1) complete section solution of continuity and separation of the proximal and distal portions to such a degree that spontaneous recovery cannot occur; (2) partial section; (3) transient lesions, the nerve has suffered temporary "concussion" and can recover spontaneously and (4) the nerve is being subjected to continued compression which, if left untreated, will inhibit recovery and may ultimately lead to "section."

"Neural" compression" is suggested as a convenient short name for this syndrome.

No special category has been given to traction lesions. To do so would produce a mixed classification based partly on the mechanism of injury and partly on the condition of the nerve. Traction injuries may produce any of the four conditions mentioned, and so may any of the other causal agents, stab wounds, bullet wounds, cervical ribs, fracture, or cubitus valgus. In the early stages it may be hard to assign a peripheral nerve lesion to its proper class except on grounds of probability, such as that "Saturday-night paralysis" is usually transient and that cuts with glass are usually complete or partial sections. At the end of three to four months' efficient treatment a nerve which has not recovered should be considered to be in one of the last three categories and should be explored so that the appropriate treatment can be given.

Significance of Changes in the Electrocardiogram After Electrically Induced Convulsions in Man. M. D. Altschule; W. M. Sulzbach, and K. J. Tillotson.

Arch. Neurol. & Psych. **58**:716 (Dec.) 1947.

The electrocardiogram after electrically induced convulsive seizures often shows evidence of a considerable increase in tone of the vagus innervation of the heart. The increase in vagal activity usually does not induce sufficient cardiac inhibition to cause more than transitory bradycardia; the systemic circulation is not slowed. Other electrocardiographic changes associated with variations in respiration and circulation after electroshock therapy are also noted.

Mycotic Vulvovaginitis and Vaginal Fungi. A Report of 280 Patients. Claudius P. Jones; Bayard Carter; Walter L. Thomas; R. A. Ross, and R. N. Creadick.

Am. J. Obst. & Gynec. **54**:738 (Nov.) 1947.

Treatment of these infections have included x-ray and ultraviolet light, various types of therapy in the form of douches, powders and salves. One of the greatest problems in the treatment of vulvovaginal mycotic infection is the avoidance of overtreatment, and the ability to determine where the fungus infection ends and the x-ray or chemical vulvovaginitis begins.

Plantar Warts. A Plea for Rational Treatment. C. R. McLaughlin.

Lancet **254**:168 (Jan. 31) 1948.

The dangers of treating plantar warts with x-rays and radium are discussed. A combination of curettage and diathermy is recommended as a safe and efficient alternative.

Some Practical Consideration in the Management of Arthritis. Joseph Lee Hollander.

West Virginia M. J. **44**:1 (Jan.) 1948.

Since rheumatoid arthritis is the most disabling of the commoner forms of arthritis, the discussion of management of arthritis will be concerned mainly with this form of joint disease. Many of the general principles of management apply to other forms of joint disease as well.

Since we realize that rheumatoid arthritis is a systemic disease, it is important that the patient rest the entire body as well as the joints involved. Rest should be mental as well as physical. The amount of physical rest a patient requires varies considerably and must be determined individually.

Many patients with rheumatoid arthritis are undernourished and underweight. It has been noted that a gain in weight often is one of the first signs of improvement.

Many patients have anemias which are treated specifically with iron, liver, and/or transfusions. At times, transfusions are given even in the absence of anemia, since an apparently non-specific, beneficial, occasionally dramatic effect sometimes follows such therapy.

The author does not believe that rheumatoid arthritis often, if ever, is cured by the removal of a so-called focus of infection.

By careful observation and anticipation of events, irreversible changes in these structures are often prevented by the use of physical therapy and orthopedic measures. It is known that muscles around diseased joints undergo atrophy, partly due to disuse, but also due to the disease itself. Therefore, heat and massage should be given daily and active and passive exercise must be done many times a day. Inflamed joints require adequate rest, but complete rest of a joint often results in permanent stiffness; thus, a fine balance between rest and regular exercises must be maintained.

An Anatomical Basis for Alterations in Quality of Pin Sensibility. G. Weddell; D. C. Sinclair, and W. H. Feindel.

J. Neurophysiol. **11**:99 (Mar.) 1948.

Biopsies were taken after sensory testing of the areas concerned, from a series of patients with either cutaneous scars of areas of partially denervated skin. It was found that in every patient in whom pain of an unpleasant quality could be elicited by means of a needle-prick, the underlying nerve nets and terminals subserving pain were isolated from their neighbors. Conversely, in no patient where this isolation was not found microscopically could pain of unpleasant quality be produced. It was also found that alterations in the quality of pain sensibility were not correlated with the presence of morphologically abnormal pain endings. Such endings were, however, associated with disturbances of the threshold of pain sensibility.

Further evidence concerning the association of pain of unpleasant quality with a reduction in the normal peripheral pattern of innervation was obtained from a number of compression experiments on the normal arm. It is considered that this association is a significant one, and that the occurrence of "over-reaction" to painful stimuli in various clinical conditions is caused by a re-

duction in the normal pattern of impulses presented to consciousness. The suggestions put forward by previous workers have been discussed in the light of this hypothesis.

The After-Treatment of Anterior Poliomyelitis. H. J. Seddon.

Practitioner **160**:176 (Mar.) 1948.

Unless there is a myositis in poliomyelitis the only change that takes place in muscle is the atrophy consequent on denervation. It must be emphasized that the process is an atrophy and not a degeneration. The affected muscles do not require the careful protection such as might be needed if they were the seat of some profound pathologic disturbance. If there is no hope of axonal regeneration there is no point in paying special attention to denervated muscles; those in which recovery occurs have retained their neuronal connexions and, apart from being out of action temporarily, are, so far as we know, in no way abnormal. Thus, apart from avoiding overstretching, which is harmful even to a normal muscle, no special mechanical treatment is required and it is to be hoped that the rigid immobilization which has been a feature of the treatment of poliomyelitis in many centers will soon become obsolete.

It has been shown that stimulation of denervated muscle with currents of long duration (galvanism) is of considerable value in preventing atrophy and it should be used routinely in the treatment of peripheral nerve injuries. However, since there is no proof that regeneration of axons plays any significant part in the process of recovery from poliomyelitis, there is no clear indication for employing electrical stimulation at any stage.

It must at once be confessed that there has been no recent advance in the treatment of the effects of paralytic poliomyelitis; all the same it is important to know when and for how long, to apply the few methods of relief that we have at our disposal.

Once the extent of the paralysis is known the treatment can be planned with precision.

In the prevention of faulty posture and overstretching of affected muscles the aim should be, not immobilization, but simply the prevention of postures that might lead to contracture, and of a normal tension on paralyzed muscles.

As soon as the patient is afebrile and free from severe pain the paralyzed parts should be put through a full range of movement every day, if possible twice a day.

The value of heat for the relief of pain in poliomyelitis has been recognized for over a generation. Massage is of use in getting rid of edema in an extensively paralyzed limb, but elevation of the part and frequent passive movements are equally, if not more, important.

Considerable recovery often occurs spontaneously, but there is no doubt that in the more severe cases much good can be achieved by re-education of muscles. The physical therapist will have three aims, the relative importance of each varying considerably from case to case. Underwater exercises are exceedingly valuable, especially in the treatment of the muscles of the trunk and lower limb.

A serious difficulty is to know when to begin and when to stop a course of treatment which is always time-consuming, tedious and expensive. The best indicator is the appearance of signs of spontaneous recovery; this must show that the inflammatory changes are subsiding, and one need then have no hesitation in starting gentle exercises. The duration of treatment must depend entirely on the progress revealed by serial muscle charts. Any muscle or segment of the body that remains completely paralyzed after four months must be written off as a loss. Occasionally, feeble power returns in such muscles towards the end of the first year or even a little later, but is hardly such as to warrant the retention of the patient in hospital and the intensive efforts of a physical therapist. Established reaction of degeneration is a most unfavorable sign, and such observations as we have been able to make during the recent epidemic suggest that electrical examination is a reliable guide to prognosis.

Retinal-Nerve Interval in the Grasshopper. Verner J. Wulff, and Theodore L. Jahn.

J. Neurophysiol. **11**:117 (Mar.) 1948.

The stellar nerves (giant axona) of Squid were exposed to two anticholinesterases, DFP and eserine. The concentration of these compounds in the axoplasm at the time of abolition of conduction was determined.

On exposure of the nerves to DFP in two concentrations, 2.2 and 5.5 x 10^{-3}M, the inside concentration was found to be less than 0.5 per cent of that on the outside. The order of magnitude of concentration in the axoplasm was 10^{-5} to 10^{-6}M. It varied in four cases from 1.3 to 8.4 x 10^{-6}M, in the fifth being 3 x 10^{-5}M.

Upon exposing the nerves to eserine in concentrations varying from 2 x 10^{-2}M to 1 x 10^{-3}M, the inside concentration was much higher than in the case of DFP at the time when the action potential had disappeared. When the eserine concentration outside was 1 x 10^{-3}M, that in the axoplasm was about the same. With higher outside concentrations the eserine inside increased slightly, but not proportionally.

The experiments show that the concentration of a compound applied externally to a nerve fiber does not indicate the concentration at the site of action. Some of the many factors determining the effectiveness of a compound applied externally are discussed.

Recent claims that conduction is possible in complete absence of cholinesterase activity have been reexamined. It was found that the data stem from the use of inadequate technic.